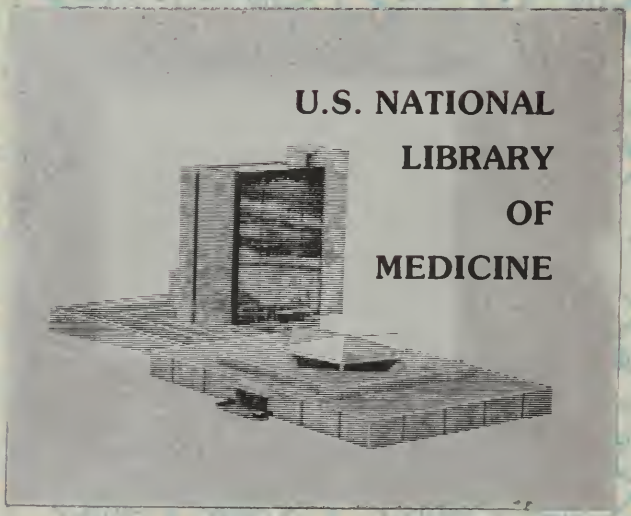
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A NEW CLASSIFICATION

OF THE

MOTOR ANOMALIES OF THE EYE

BASED UPON PHYSIOLOGICAL PRINCIPLES.

TOGETHER WITH THEIR

SYMPTOMS, DIAGNOSIS

AND

TREATMENT.

THE PRIZE ESSAY OF THE ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK, FOR 1896.

BY

ALEXANDER DUANE, M. D.,

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PREFACE.

The following brochure respresents the result of some ten years' labor and study expended upon the subject of muscular anomalies. Whatever merit it may have is due to the fact that it stands for original investigation in a field still full of difficulties and obscurities. The author's clinical experience has convinced him that the classifications propounded furnishes an adequate working basis for the diagnosis of these conditions. And his experience as a teacher at the Ophthalmic and Aural Institue has led him to believe that the principles here laid down, and the means and methods of examination here recommended, have been found by others also to be both intelligible and practicable. Many of these principles and methods have been enunciated in lectures given to successive classes of practitioners, and have been demonstrated in their practical application upon patients before the same gentlemen; the auhor's constant attempt being to present clearly and in a way suited to general comprehension the rules for the diagnosis and management of the muscular anomalies. That this attempt was not unsuccessful he has had some reason to believe from the assurances of those that he has taught; and it is, therefore, with the hope that the result of his work may be useful to others as well that he now offers it to the public.

ALEXANDER DUANE, M. D.

49 East Thirtieth St., New York.
April 29, 1897.

ERRATUM.

Page 23, line 11: For "22," read "$2^1/_2$."

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ILLUSTRATED.

Introduction.—Sketch of previous classifications.—Development of the idea of an etiological, as opposed to a simple anatomical classification.

I. **Nature of the problems that have to be solved** in undertaking a physiological classification.

II. **The Movements of the Normal Eye.**—Actions of the individual muscles.—Movements of each eye individually and the muscles by which they are performed.—Amount of these movements.—Field of fixation.—Author's experiments.—Power of the individual muscles.—Coördinated movements of the two eyes.—Table of associated parallel movements and the muscles producing them.—Associated antagonists.—Field of binocular single vision and of binocular fixation.—Author's experiments.—Movements of convergence.—Power of convergence.—Convergence near-point.—Prism-convergence.—Movements of divergence.—Nature of divergence action.—Movements of sursumvergence.—Rotation movements.—Appendix.—Diagrammatic representation of the movements of the eye.

III. **The Tests Employed and Their Significance.**—Object of the tests.—Tests for binocular distant fixation.—Inspection.—Fixation and diplopia tests.—Equilibrium tests —Screen test.—Parallax test.—Tests for associated parallel movements.—Tests for convergence.—Tests for divergence.—Tests for sursumvergence.—Way in which the tests are applied in practice.

INTRODUCTION.

The nomenclature and classification of the muscular anomalies of the eye have been passing through the same stages that have been noted in the evolution of the nosology of other parts of the body. In the progress of our knowledge in regard to any given

set of ailments, the first classification has always been based upon that which first strikes the eye of the observer, namely, the outward appearances and symptoms. Thus, many cases of renal disease were first classified as dropsy, and dropsy formerly figured among physicians, as it does still among the laity, as a substantive disease, and to be treated as such. But, as medical science progressed, and the underlying causes of disease were more and more brought to light, it became evident that dropsy is a symptom only and to be treated as a symptom, and that the principles of a rational pathology require us to search for the causes of the dropsy and to institute treatment addressed to the removal of these causes rather than to the direct relief of the dropsy itself. In this way a pathological classification is gradually substituted for one that is purely symptomatic, and a scientific, casual treatment for one that is empirical, or based solely on the appearances presented.

This conception of disease as a symptom of a pathological process, and the consequent conviction that our therapeusis must be based ultimately upon an etiological foundation, could not have developed, or at all events, could not have become anything more than a plausible theory, barren of practical application, were it not constantly fortified by a steady increase in the number and precision of our means of diagnosis. In this way only can we make those fine discriminations between symptoms that enable us to form accurate inferences as to the diverse origin of phenomena which, to a cursory observation, seem identical. For example, our knowledge of the symptomatic nature of dropsy and of the necessity of treating it from an etiological standpoint, however true it might be, would be a theory only, unprovable and practically inapplicable, were it not for the refined means we now possess for examining the chest and abdomen and for analyzing the urine. These diagnostic means have enabled us to convert theory into fact, and to redeem our treatment from the charge of empiricism.

A similar process of evolution has taken place in regard to the motor anomalies of the eye. These were formerly (and to a great extent still are) classified simply according to the appearances presented, i. e., as inward, outward, upward, or downward deviations. And, to recur to our former illustration, just as dropsy used to be treated as dropsy, regardless of whether it was due to renal, cardiac, or hepatic disease, so an inward squint was (and often still is) treated simply, as a squint, quite without reference to its origin. The results in both cases have often been disappointing.

The first great step in advance was taken by Donders, when he demonstrated the frequent connection and apparent causal relation between strabismus convergens and hypermetropia, and between strabismus divergens and myopia. And, while subsequent writers have doubtless gone too far in asserting the universal application of his deductions, the service that he himself did to rational therapeutics by indicating one large class of cases in which a strictly causal treatment relieves the symptoms, can scarcely be overestimated.

Another exceedingly important advance made was in the discovery of the insufficiencies and of the methods of estimating them. These latter, devised by V. Graefe, remain to-day among the chief means of discriminating between the various motor anomalies; and their invention, as in the case of all new methods of precision, opened the way to still further discoveries.

The diagnosis and symptomatology of paralytic deviations was also worked out by the same author, and in a manner so admirably complete, that little remained for his successors to do except to gather statistics in regard to the precise etiology of these interesting affections.

V. Graefe had pointed out the relation between insufficiency and squint, but since his time this relation has been largely overlooked, the consequence being that the two conditions have often been classed as categorically distinct, and distinct principles of treatment have been applied to them. This arbitrary separation of two things really the same has had a retarding influence upon the development of our knowledge of muscular anomalies.

It became, after awhile, apparent that the very term insufficiency was a defective one, implying, as it did, a casual relation, which was by no means always present. Hence, Stevens in this country proposed a system of classification in which the term insufficiency was replaced by *heterophoria*, and the term strabismus by *heterotropia*, the former indicating a tendency to deviation, which tendency is habitually overcome, and the latter a deviation which is more or less constantly present. The further use of the prefixes Eso-, Exo-, and Hyper-, served to differentiate between deviations or tendencies inward (convergent deviation), outward (divergent deviation), and upward (or, more properly, divergence in a vertical plane). This classification is extremely convenient and has been generally accepted, at least in America. Furthermore, the instruments of precision, which the same author devised for measuring the various deviations, are doubtless the best extant, and have greatly facilitated the recognition and differentiation of the anomalies in question.

Stevens also did service in pointing out that many cases of so-called insufficiency, or heterophoria, were really low degrees of squint—a fact which, as already stated, has been too much overlooked.

He further called particular attention to the importance and frequency of the vertical deviations which had received (and, in fact, still receive) too little notice; the fact being that their recognition and correction are very necessary features in the management of many cases of muscular trouble.

Stevens' classification, however, is open to the serious objection that it reinstates the idea of grouping deviations according to their anatomical characters and puts the etiological element in the background. That is, it aims to classify motor disorders according to their outward, visible characters, and not according to their cause. In this way, though admirable in other respects, it would seem to be a distinct step backwards. Thus, while the term insufficiency is faulty, because it restricts too narrowly our conception of the possible etiology of a given motor affection, the term heterophoria is objectionable, because it throws away the etiological idea altogether, and once more directs our attention simply to the appearances present, i. e., to the fact that the eye in a certain case deviates up, down, out, or in. To be sure, this has its advantages in that there are many cases in which we cannot say at once, or even after considerable testing, what the true condition underlying these appearances is, and we have, therefore, to content ourselves with a provisional—i. e., an anatomical—diagnosis. But we should always feel that such a diagnosis is provisional, and that a really satisfactory diagnosis should express the cause of the deviation as well as its character. That is, the statement that in a given case so many degrees of esophoria were found should be regarded as only an incomplete presentation of the facts; the final diagnosis not being reached until we have determined the cause of the esophoria, i. e., have made out which one of the many and complex functions of the neuro-muscular apparatus of the eye is deranged. In the meantime, the terms that Stevens has devised are very convenient for purposes of record and for indicating the provisional diagnosis.

The next step naturally left to take—the classification, namely, of the motor affections of the eye upon an etiological basis—has been but partially made. Apart from Donder's researches before mentioned, and V. Graefe's demonstrations of the characters

displayed by paretic squint, the main contributions to the subject have been A. Graefe's description of convergence-insufficiency, Landolt's observations upon the comparative effect and relative value of advancement and tenotomy in the treatment of various kinds of strabismus, and some of Stevens' recent papers.

Nowhere, however, have the observations bearing upon this point been properly co-ordinated, so as to form a complete classification of all the different anomalies, founded upon a strictly etiological and physiological basis. To do this, at least in part, is the object of the present essay, which is based almost exclusively upon observations made by the author himself during the past ten years. The subject has seemed to him to be one not only of scientific interest, but also of great practical importance, since, as experience has shown, our plans of treatment are necessarily largely modified by our notions of the real nature and causes of the condition that we are called upon to correct. Moreover, the cases themselves are very numerous; their symptoms, subjective and objective, are multiform and complex; while the results of treatment, which are often brilliant, are often also disappointing, owing, no doubt, frequently, to our ignorance of the exact nature of the case before us. It has seemed proper, therefore, to enter into considerable detail in trying to determine what should be the proper classification of such cases, and what precisely are the differential marks by which they can be distinguished one from the other.

I.

NATURE OF THE PROBLEM.

The task that confronts us when we attempt to make any classification of the kind now essayed is that of framing a scheme by reference to which the following problem may, in most instances, at least, be resolved: *Given a case with a certain train of symptoms, to determine the ultimate cause of these symptoms, i. e., the part or function that is primarily deranged, and the manner of its derangement.* The solution of this problem obviously presupposes, first, an accurate knowledge of the normal state of the various parts and functions that may be involved, and, second, a consideration of the different means we have for determining whether each part or function is actually normal or not. Thus, in the case of deformities about the hip, a proper classification, i. e., one which tells us whether any given deformity is due to a fracture, to a dorsal dislocation, or to hip-disease, is possible only after we have become thoroughly acquainted with the normal relations and movements of the hip-joint, and with the means at our

command for appreciating the various deviations from the normal and their significance. Applying this principle to the eye, we may say that the solution of our problem is contained in the answers to the following questions:

(1). What are the different normal functions of the neuromuscular apparatus of the eye, and what is their anatomical seat?

(2). In what way is it possible for these functions to be deranged?

(3). What means have we for telling whether any special function is deranged or not, and, if so, what the nature of the derangement is?

(4). What are the conditions actually met with in practice, and how do they correspond to the scheme that we have framed?

The consideration of these questions we will now take up in the order named.

II.

MOVEMENTS OF THE NORMAL EYE.

The functions concerned in the group of cases under consideration comprise (*a*) the *actions of the individual muscles* that are inserted into the eyeball; (*b*) the *movements possible to each eye separately* through the individual or concerted working of these muscles and (*c*), the character and extent of the *movements actually performed by the two eyes when acting together.*

The **actions of the individual muscles** are best exhibited in tabular form, as follows:

Muscle	Field of action limited to	Moves eye laterally.	Rotates upper end of vertical meridian of cornea (torsion effect.)	Lateral and torsional effects increasing as eye is	Lateral & torsion effects diminishing to zero, as eye is	Moves eye vertically	Vertical action increasing as eye is	Vertical action diminishing as eye is
External Rectus.	Outer half of field of fixation	Out	No action			No action		
Internal Rectus.	Inner half of field of fixation	In	No action			No action		
Superior Rectus.	Upper half of field of fixation	In	In	Adducted.	Abducted.	Up	Abducted,	Adducted
Inferior Rectus.	Lower half of field of fixation	In	Out	Adducted.	Abducted.	Down	Abducted.	Adducted
Superior Oblique	Lower half of field of fixation	Out	In	Abducted.	Adducted.	Down	Adducted.	Abducted
Inferior Oblique	Upper half of field of fixation	Out	Out	Abducted	Adducted.	Up	Adducted.	Abducted

It will be seen from the foregoing table that under ordinary conditions the only two muscles which precisely counteract one another's action, or which are, in the language of physiology, direct antagonists, are the external and internal recti.

The superior and inferior recti, for example, are only partially antagonistic, for, while respectively elevating and depressing the eye, so as to be directly opposed to each other in this regard, and while their action in rotating the vertical meridian of the cornea is also precisely opposite, they both adduct the eye. Hence, when acting together they will reinforce the internal rectus, and this action will be most strongly pronounced when the eye is already markedly adducted, i. e., under conditions in which the internal rectus is working at some mechanical disadvantage. The like is true of the combined action of the two obliques, which neutralize each other as far as rotation of the vertical meridian and as far as elevation and depression are concerned, but work together in producing abduction and hence assist the external rectus, especially when the eye is already strongly abducted. On the other hand, when the eye is strongly adducted, the lateral action of the two obliques falls away, and these two muscles act simply to elevate and depress the eye respectively. In this position, therefore, they do neutralize each other perfectly, and are direct antagonists. The same is true of the superior and inferior recti when the eye is abducted. For further remarks upon this subject, see the appendix to this chapter.

Almost every movement that the eye can make *requires the combined action of at least two* of the ocular muscles. Thus to lift the eye straight upwards, we must use both the superior rectus and the inferior oblique.

The superior rectus, acting by itself, would carry the eye inward as well as upward, and would rotate the vertical meridian of the cornea inward. So, too, the inferior oblique, acting by itself, would tend to abduct the eye and rotate the vertical meridian outward. Neither, therefore, alone will carry the eye straight upward, but the two acting together, will neutralize each other as far as their lateral working and their effect upon the vertical meridian are concerned, and consequently the eye rises vertically, without swerving to the right or left, and without any deflection of its vertical meridian.

It is probable that the external and internal recti assist in maintaining the strict verticality of this movement, their simultaneous contraction steadying the eye and preventing it from swerving. In this case, therefore, at least two, and probably four muscles, are concerned in the movement.

Similarly, depression of the eye is always accomplished by the conjoint action of the inferior rectus and the superior oblique, which neutralize each other to a greater or less extent, as far as their lateral working and their effect upon the vertical meridian are concerned, but which assist each other in carrying the eye downward. Here, too, probably the external and internal recti come into play as steadying and supporting factors.

Even in so simple a movement as that of abduction, which might be performed by a single muscle, it is probable that at least two

(i. e., both obliques), or even four other muscles (i. e., all except the internus), take part either in reinforcing the action, or in steadying the eye and rendering the movement uniform. See Appendix.

The movements of the individual ocular muscles are, as is well known, *presided over by more or less discrete nuclei* scattered along the walls of the third ventricle, aqueduct of Sylvius, and fourth ventricle; but the precise method in which these are arranged and inter-connected has not yet been sufficiently determined.

The Movements Possible to the Eye through the co-ordinated action of its six muscles comprise rotations in every conceivable plane, the eye being capable of moving from the primary position directly to any secondary position, and from the latter again to any other secondary position, and in so doing may take either a direct or a sinuous course.* The typical direct movements from the primary position, together with the muscles concerned in the production of these movements, are shown in the following table:

*This may be proved by making two fine dots upon a card, so close together that unless very accurately fixed (i. e., if seen ever so slightly in diffusion circles), they will blend into one, and then moving the card slowly in all directions before the eye, the head remaining fixed. However the card is moved, the two dots will remain distinct, thus showing that the eye follows them in all their movements.

Direction of movement from primary position.	Muscles Concerned.	Move Eye Laterally*	Move Eye Vertically*	Rotate Upper End of Vertical Meridan of Cornea*	Resultant Effect Upon Eye.
Out (Abduction)	External Rectus	Out	No action.	No action.	Eye carried out mainly by action of external rectus assisted by the two obliques; the effect of the latter being the greater the more the eye is abducted. The opposing (adducting) action of the superior and inferior recti also diminishes as the eye is abducted. The eye is steadied and its vertical meridian kept vertical by the traction exerted by the superior and inferior recti and the two obliques.
	Synergists.				
	Superior Oblique	Out — Action increasing the further the eye is abducted,	[Down] — Action equal and opposite; decreasing the more the eye is abducted.	[In] — Actions equal and opposite; increasing the more the eye is abducted.	
	Inferior Oblique	Out — Action increasing the further the eye is abducted,	[Up] — Action equal and opposite; decreasing the more the eye is abducted.	[Out] — Actions equal and opposite; increasing the more the eye is abducted.	
	Opponents.				
	Superior Rectus.	[In] — Actions slight and decreasing the more the eye is abducted.	[Up] — Actions equal and opposite; increasing the more the eye is abducted.	[In] — Actions equal and opposite; decreasing the more the eye is abducted.	
	Inferior Rectus.	[In] — Actions slight and decreasing the more the eye is abducted.	[Down] — Actions equal and opposite; increasing the more the eye is abducted.	[Out] — Actions equal and opposite; decreasing the more the eye is abducted.	
In (Adduction.)	Internal Rectus	In	No action	No action.	Eye carried in mainly by internal rectus, assisted by the superior and inferior recti: the effect of the latter being greater, the more the eye is adducted. The opposing (abducting) action of the two obliques diminishes as the eye is adducted. The eye is steadied and its vertical meridian kept vertical by the counterpoising action of the two obliques and the superior and inferior recti.
	Synergists.				
	Superior Rectus	In — Action increasing the more the eye is adducted.	[Up] — Actions equal and opposite; decreasing as the eye is adducted.	[In] — Actions equal and opposite; increasing as the eye is adducted.	
	Inferior Rectus	In — Action increasing the more the eye is adducted.	[Down] — Actions equal and opposite; decreasing as the eye is adducted.	[Out] — Actions equal and opposite; increasing as the eye is adducted.	
	Opponents.				
	Superior Oblique	[Out] — Actions slight and diminishing as the eye is adducted,	Down — Action equal and opposite; increasing as the eye is adducted	[In] — Actions equal and opposite; diminishing as eye is adducted.	
	Inferior Oblique	[Out] — Actions slight and diminishing as the eye is adducted,	[Up] — Action equal and opposite; increasing as the eye is adducted	[Out] — Actions equal and opposite; diminishing as eye is adducted.	

* Movements that are completely neutralized by the action of opposing muscles are placed in brackets.

Direction of Movem't from Primary Position.	Muscles Concerned.	Move Eye Laterally	Move Eye Vertically	Rotate Upper End of Vertical Meridian of Cornea	Resultant Effect Upon the Eye
Up (Sursumduction)	Superior Rectus	[In] Movements equal and opposite.	Up.	[In] Actions equal and opposite.	Eye carried straight up; vertical meridian remains vertical. The counterpoising action of the internal and external recti serves to steady eye and keep it in the vertical line.
	Inferior Oblique	[Out] Movements equal and opposite.	Up.	[Out] Actions equal and opposite.	
	Synergists.				
	External Rectus	[Out] Actions equal and opposite.	No action	No action	
	Internal Rectus	[In] Actions equal and opposite.	No action	No action	
Diagonally Up and Out.	Superior Rectus	[In]; Action slight and diminishing as eye is abducted.	Up, Action marked and increasiug as eye is abducted.	[In]; Action slight and diminishing as eye is abducted.	Eye carried *up* mainly by superior rectus, the elevatiug action of this muscle increasiug and that of the inferior obllque dimiuishing as the eye is abducted. Eye carried *out* mainly by external rectus, assisted by inferior oblique, especially when abduction is marked.
	Inferior Oblique	Out; Action increasing as eye is abducted.	Up; Action slight and decreasing as eye is abducted.	Out: Action marked and increasiug as eye is abducted.	
	External Rectus	Out.	No action.	No actiou.	Vertical meridian rotated *out*.
Diagonally Up and In.	Superior Rectus	In: Action increases as eye is carried inward.	Up; Action slight and decreasing as eye is adducted; finally 0.	In; Action increases as eye is carried in.	Eye carried *up* mainly by the inferior oblique, the elevating action of this muscle increasiug and that of the superior rectus decreasing as the eye is adducted. Eye carried *in* by the internal rectus, assisted, especially when adduction is marked, by the superior rectus, Vertical meridian rotated *in*.
	Inferior Oblique	[Out]; Action slight and decreasing as eye is carried in.	Up; Action marked and increasing as eye is carried in.	[Out] Action slight and decreasing as eye is carried in.	
	Internal Rectus.	In.	No action.	No action.	
Down (Deorsumduction)	Inferior Rectus	[In] Actions equal and opposite.	Down.	[Out] Action equal and opposite.	The eye carried straight down, vertical meridian remaiuing vertical. Exterual and internal recti by their countertraction serve to steady eye and keep it in the vertical line.
	Superior Oblique.	[Out]	Down.	[In]	
	Synergists				
	External Rectus.	[Out] Actions equal and opposite.	No action.	No action.	
	Internal Rectus.	[In]	No action.	No action.	

Direction of Movement from Primary Position	Muscles Concerned.	Move Eye Laterally.	Move Eye Vertically.	Rotate Upper End of Vertical Meridian of Cornea.	Resultant Effect Upon Eye.
Diagonally Down and Out	Inferior Rectus.	[In]; Action slight and diminishing as eye is abducted.	Down; Action marked and increasing as eye is abducted.	[Out]; Action slight and diminishing as eye is abducted.	The eye carried *down* mainly by inferior rectus, the depressing action of this muscle increasing and that of the superior oblique decreasing as the eye is abducted. Eye carried *out* mainly by external rectus, assisted especially in extreme abduction, by the superior oblique. Vertical meridian rotated *in*.
	Superior. Oblique	Out; Action increasing as eye is abducted.	Down; Action slight and diminishing as eye is abducten.	In; Action increasing as eye is abducted.	
	External Rectus.	Out	No action	No action.	
Diagonally Down and In.	Superior Oblique.	[Out;] Action slight and diminishing es eye is adducted.	Down; Action marked and increasing as eye is carried inward.	[In;] Action slight and diminishing as eye is adducted.	The eye carried *down* mainly by the superior oblique, the depressing action of this muscle increasing and that of the inferior rectus diminishing as the eye is adducted. Eye carried *in* mainly by the internal rectus, assisted especially in extreme adduction by the inferior rectus; vertical meridian rotated *out*.
	Inferior Rectus	In; Action increasing as eye is adducted.	Down; Action slight and diminishing as eye is adducted.	Out; Action increasing as eye is adducted.	
	Internal Rectus.	In.	No action.	No action.	

The *amount that the eye can move* in each one of the directions specified may be determined experimentally by placing the subject experimented upon with his eyes in the primary position and directed at an object whose recognition implies accurate fixation, (e. g.) a fine double dot on a card, and then moving the object in the given direction, requiring the patient at the same time to follow it with his eyes, but not with his head. The moment when he ceases to follow it will be evidenced objectively by the perceptible wavering of the eye, which hitherto had steadily followed the object, and subjectively by the fact that the object itself becomes confused and no longer recognizable. Then the arc through which the eye has rotated in passing from the primary to the terminal position may be measured either roughly with the eye, or accurately by some form of perimeter.*

*This may also be accomplished by Stevens' tropometer or some similar instrument which measures the rotation of the eye by measuring the arc traversed by the corneal reflex.

By ascertaining the limits of movement in all directions, we define the boundaries of the *field of fixation* i. e., of the entire space through which the visual line can be carried without moving the head.

The measurement of the field of fixation in any given case requires that the patient under examination shall, in each excursion that he makes with his eyes, put forth the maximum effort of which he is capable. This he will frequently fail to do, thereby making the field appear incomplete. It is only by making several examinations and taking the maximum of all the measurements, that we arrive at a perfectly reliable result, i. e., one which shows the full extent of excursion of which the eye is capable. The discrepancies thus obtained in repeated examinations are well shown in cases 1, 5, and 6, of the following table, which is constructed from observations of my own, made upon normal eyes with the perimeter, and using the fine double dot as a test-object.

RIGHT EYES*

Case.	Up	Up & Out	Up & In	Out	In	Down	Down & Out	Down & In
1 1st exam.	40	35	40	35	40	70	35	25
" 2d exam.	38	45	40	48	48	42	55	50
2	40	50	40			45	60	60
3	50	45	55	55	45	60	65	55
4 Right eye not examined.								
5 1st exam.	40	48	(50)	60	(50)	62	65	(50)
" 2d exam.	40	48	(55)	50	(55)	(62)	50	(60)
" 3rd exam.	(30)	42	(42)	50	(45)	60	65	(60)
6 1st exam.	32	45	32	52	52		65	50
" 2d exam.	38		50					
7	32	35	35	55	52	72	65	(62)
8	44	55	55	60	60	62	70	(65)
9	50	50	60	52	(60)	80	(70)	60
10	40	40	42	50	42	45	50	40
11 1st exam.	47	50	50	40	63	63	67	55
12 Right eye not examined.								
13	45	55	(55)	50	60	55	52	(53)
14 1st exam.	52	60	55	52	50	62	60	55
" 2d exam.	40	50	(45)	65	(50)	65	80	(50)
15	(50)	45	52	48	56	62	58	(60)
16 1st exam.	52	50	55	48	56	60	60	(50)
17 1st exam.	40	38	50	40	(45)	68	(65)	(50)
" 2d exam.	47	52	52	48	(48)	60	(40)	60
18	60	70	62	75	60	65	73	60

*Figures enclosed in parentheses mean that at this point the test object disappeared from view behind some projecting part of the face, but at the time of disappearance was still within the field of fixation.

LEFT EYES.

Case	Up	Up & Out	Up & In	Out	In	Down	Down & Out	Down & In
1 1st exam.	50	40	45	35	45	70	60	50
" 2d exam.	42	40	55	48	60	70	50	60
2	40	50	40			60	50	60
3	35	40	45	55	50	65		75
4	40	38	46	48	40	35	38	32
5 1st exam.	40	52	42	53	[48]	65	70	[50]
" 2d exam.	43	50	[44]	52	48	[68]	62	[48]
" 3rd exam	42	50	40	48	[50]	65	60	[50]
6 Left eye not examined.								
" " " " "								
7	35	42	45	55	52	70	65	60
8 Left eye not examined.								
9	55	60	60	58	60	72	75	[40]
10 Left eye not examined.								
11 1st exam.	50	45	55	47	60	65	68	60
12	50	60	50	50	[60]	70	62	[40]
13	[40]	55	50	50	[50]	60	75	
14 1st exam.	46	60	55	55	45	72	72	60
" 2d exam.								
15	48	50	50	56	55	65	60	[60]
16 1s exam.	48	55	52	55	52	60	55	52
17 1st exam.	40	42	[50]	40	62	70	60	[52]
18 2d exam.	44	50	50	48	[50]	62	62	[40]
"	58	71	71	72	62	72	76	68

The average of the observations above tabulated gives rather a larger field of fixation than has been obtained by other experimenters. Thus, Landolt's figures, while showing a close agreement for excursions in the upper field, are appreciably less for movements downward (about 50° in looking down, 38° in looking down and out, and 47° in looking down and in). On the other hand, the experiments of Schuurmann and Donders give the range of downward excursion as 57°, which is somewhat less than those that were found by me, but the upward excursion as only 34°. This latter figure certainly seems too small, in view of the fact that in but one of my cases was the range as low as this, and that in nearly all the others it fluctuated between 40° and 50°.*

From the range of excursion of the eye in various directions, we can form a tolerably close estimate of the *amount of work that each muscle does* in moving the eye.

For example, when the eye is abducted 30°-35°, its movement upwards is effected solely by the superior rectus, and, moreover, the latter is then at its maximum as an elevator. Hence, to determine the maximum elevating power of this muscle, we have only to measure the range of excursion upwards that the eye can make when abducted to this extent. Similarly, the range of excursion downwards, when the eye is abducted 30°, measures the maximum depressing power of the inferior rectus. Making use of the results

*Certainly the value of 20° found for the range of upward excursion by Hering (cited in Graefe-Saemisch) seems excessively small. It is not unlikely that here there was a pathological condition present, such as an insufficiency of one or both elevators—a phenomenon not infrequent.

already tabulated,* we find for the *maximum elevating power of the superior rectus* a value of 30°, and for the *maximum depressing power of the inferior rectus*, a mean value of 35°-40°.

The maximum elevating power of the inferior oblique and the maximum depressing power of the superior oblique, are not so readily determined, as the eye can hardly be so far abducted as to enable these muscles to work to the greatest advantage, and at the same time do away altogether with the vertical action of the superior and inferior recti. It would appear, however, that the *maximum vertical effect exerted by the obliques*, does not differ materially from that exerted by the straight muscles; only the effect of the latter in the positions ordinarily assumed by the eye is rather more pronounced.

The *maximum rotating effect* of the superior rectus upon the vertical meridian (torsion-effect, swivel-movement) will be ascertained by determining the amount of deflection of the vertical meridian when the eye is directed far up and in. In this situation the vertical meridian is not acted upon by the other muscles capable of

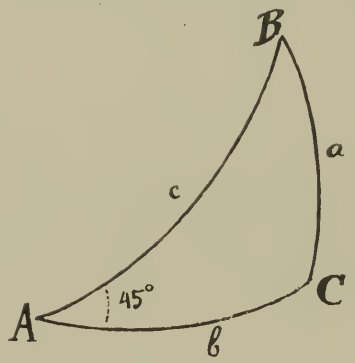

Fig. 1.

rotating it, so that the total rotation it undergoes must be ascribed to the action of the superior rectus. So, too, the rotating power of the inferior oblique, the superior oblique, and the inferior rectus is measured by the amount of tilting of the vertical meridian when the eye is directed up and out, down and out, and down and in, respectively.

The *adducting effect* of the superior and inferior recti and the *abducting effect* of the obliques are not determinable directly in the normal eye, since these actions always occur as reinforcements of the adducting and abducting actions of the internal and external recti.

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*This can be accomplished by a simple calculation, based upon the principles of spherical trigonometry. Thus if AB represents the path of the visual line in passing from the primary position obliquely up and out, BC (or a) will be the elevation and AC (or b) the outward excursion or abduction of the eye. Then Sin. a = Sin. A. Sin. c and Sin. b = cot A. tan. a. Here c = the range of excursion up and out as given by our table (= about 45°), a = 30, and b = 36°.

For the same reason, the total adduction or abduction that the eye is capable of is not a precise measure of the *maximum power of the external and internal recti*, since the latter are to a certain extent, assisted in their action by the vertical muscles, and particularly so when the eye is already carried pretty far out or in. It is probable, however, from observations in cases of paralysis, that the lateral action of the vertical muscles does not amount to more than 4° or 5° at most, so *that the abducting power of the externus* may be stated as 40°-45°, and the *adducting power of the internus* as about 50°.

Passing now to the third group of functions under consideration, namely **the character and relations of the movements performed by the two eyes when working together** we are struck by the fact that, with unimportant exceptions,* these movements are limited to those subserving binocular fixation.

Thus, in order to produce the binocular fixation of distant objects, the visual lines must be parallel. In harmony with this fact, we find that there is a whole series of movements—*associated parallel movements*—in which the visual line of one eye is kept strictly parallel with that of the other; and, moreover, the vertical meridians of the two cornæ also remain parallel, no matter how the eye is directed.

A second class of movements—*movements of convergence*—adapt the eyes for the binocular fixation of near objects.

A third sort of movement is that of *divergence in the horizontal plane*, causing the eyes to pass from the consideration of a near object to that of one more remote. In doing this, this movement, like the preceding, subserves binocular fixation; but it may to a certain limited extent, also antagonize the latter by carrying the eyes still further, i. e., from a position of parallelism to one of actual divergence.

Another kind of movement, of very limited extent, is that of *divergence in a vertical plane*, produced by the elevation of one visual line and the depression of the other. This movement, called sursumvergence (or deorsumvergence) is denoted as right or left, according as the right or left visual line is higher (or lower).

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*The exceptions are very clearly described by Helmholtz (Phys. Optik, 2d Ed., pp. 631, et seq.) He seems to me, however, to have laid too much stress upon the ability of the eye to make exceptional movements of this sort; the fact being that such movements are extremely limited and, beyond a certain point, cannot be increased by practice. It seems, therefore, that in spite of his statement to the contrary, there is some anatomical basis for the inability of the eye to make unaccustomed movements and that it is not simply a question of training.

A fifth class of movements comprise those in which the *vertical meridians of the two cornea are so rotated* as to be no longer parallel. Such a rotation occurs normally in positions of marked convergence, the vertical meridians then diverging at their upper extremities, and the amount of divergence increasing as the eyes are elevated (Meissner, Le Conte). But, apart from this physiological torsion-movement, a divergent or convergent rotation of the vertical meridians may be produced in the normal eye and demonstrated by suitable apparatus (Helmholtz).

The Associated Parallel Movements of the two eyes with the muscles concerned in their production are shown in the following tables:

Both Eyes Moveto	Muscles concerned: R. Eye	Muscles concerned: L. Eye	Move eyes laterally*	Move eyes vertically*	Rotate upper end of vertical meridians of corneæ.	Resultaut effect upon eyes.
Right (Dextroversion.)	External Rectus	Internal Rectus	To R.	No action	No action	R eye carried to R by the external rectus assisted by the obliques: L eye carried to R by iuternal rectus assisted by the superior and iufer ior recti, The other muscles act to steady the eyes and keep them in the horizoutal plane. Vertical meridiansboth remain vertical.
	Synergists					
	Superior Oblique	Inferior Rectus	To R, action increasing the more eyes are carried to R	[Down actious equal aud opposite	[To L actions equal and opposite.	
	Inferior Oblique	Superior Rectus		[Up	[To R	
	Opponents.					
	Superior Rectus	Inferior Oblique	[To L]action sli't & diminishing as eyes are carried to R.	[Up] Actions equal and opposite	[To L actions equal and opposite.	
	Inferior Rectus	Superior Oblique		[Down	[To R	
Left (Sinistroversion.)	Internal Rectus	External Rectus	To L-	No action.	No action.	R. eye carried to L. by the internal rectus, assisted by the superior and inferior recti: L. eye carried to R. by the external rectus, assisted by the obliques. The other muscles act to steady the eyes and keep them in the horizontal plane. Vertical meridians remain vertical
	Synergists.		To L action increases the more eyes are carried to L.			
	Superior Rectus	Inferior Oblique		[Up] actions equal and opposite.	[To L actions equal and opposite.	
	Inferior Rectus	Superior Oblique		[Dowu	[To R	
	Opponents.					
	Superior Oblique	Inferior Rectus	[To R] action slight & decreases the more eyes are carried to L	[Down actions equal and opposite.	[To L actions equal and opposite.	
	Inferior Oblique	Superior Rectus		[Up]	[To R	
Up [Sursumversion.]	Superior Rectus	Inferior Oblique	[To L] actions equal & opposite	Up	[To L actions equal and opposite	Both eyes carried vertically up by combined action of superior rectus and inferior oblique. External and internal recti act to steady eye and keep it in vertical plane. Vertical meridians remain vertical.
	Inferior Oblique	Superior Rectus.	[To R]	Up	[To R	
	Synergists.					
	External Rectus	Internal Rectus	[To R] actions equal & opposite	No action	No action	
	Internal Rectus	External Rectus	[To L]	" "	" "	

*Portions enclosed in brackets indicate movements which are completely neutral ized.

Both Eyes Move	Muscles concerned R Eye	Muscles concerned L Eye	Move eyes laterally	Move eyes vertically	Rotate upper ends of vertical meridians of corneæ	Resultant effects upon eyes
Up and to R.	Superior Rectus	Inferior Oblique	[To L.] Action slight and decreasiug as eyes are carried to R	Up. Action marked and increasing as eyes are carried to R.	[To L] Action slight and decreasing as eyes are carried to R	R. eye carried up by superior rectus; L. by inferior oblique. R. eye carried to R. by external rectus assisted (especiallyinextreme abduction) by inferior oblique: L. eye carried to R by internal rectus assisted by superior rectus. Both vertical meridians tilted to R.
	Inferior Oblique	Superior Rectus	To R. Actious marked & increasing as eyes are carried to R	Up. Action slight and decreasing as eyes are carried to R.	To R. Action marked & increasing as eyes are carried to R	
	Synergists.				No action.	
	External Rectus	Internal Rectus	To R.	No action.		
Up and to L.	Inferior Oblique	Superior Rectus	[To R] Action slight & decreasing as eyes are carried to L.	Up. Action marked and increasing as eyes are carried to R	[To R] Action slight & decreasing as eyes are carried to L.	R. eye carried up mainly by inferior oblique, L eye by superior rectus; R eye carried to L by internal rectus assisted by superior rectus. L eye carried to L by external rectus assisted by inferior oblique. Vertical meridians both rotated to L
	Superior Rectus	Inferior Oblique	To L. Action marked & increasing as eyes are carried to L.	Up. Action slight and decreasing as eyes are carried to L.	To L Action marked and increasing as eyes are carried to L.	
	Synergists.					
	Internal Rectus	External Rectus	To L.	No action.	No action.	
Down (Deorsum version)	Inferior Rectus	Superior Oblique	[To L } action equal & opposite	Down.	[To R } action equal & opposite	Both eyes carried vertically down by combined action of inferior rectus and superior oblique. External and internal recti act to steady eyes and keep them in the vertical plane. Vertical meridians remain vertical.
	Superior Oblique	Inferior Rectus	[To R }	Down.	[To L }	
	Synergists.					
	External Rectus	Internal Rectus	[To R } action equal & opposite	No action.	No action.	
	Internal Rectus	External Rectus	[To L }	" "	" "	

Both Eyes Move	Muscles concerned R Eye	L Eye	Move eyes laterally	Move eyes vertically	Rotate upper ends of vertical meridians of corneæ	Resultant effects upon eyes
Down and to R.	Inferior Rectus	Superior Oblique	[To L.] Action slight & decreasing as eyes are carried to R.	Down. Action marked and increasing as eyes are carried to R	[To R.] Action slight and decreasing as eyes are turned to R.	R. eye carried down mainly by iuferior rectus, R. eye by superior oblique, R. eye carried to R by external rectus assisted by superior oblique, L. by iuternal rectus assisted by inferior rectus. Both vertical meridians rotated to L.
	Superior Oblique	Inferior Rectus	To R. Actions marked & increasing as eyes are carried to R	Down. Actions slight and decreasing as eyes are carried to R.	To L. Action marked and increasing as eyes are carried to R.	
	Synergists.					
	Exterual Rectus	Internal Rectus	To R	No action.	No action.	
Down and to L.	Superior Oblique	Inferior Rectus	[To R.] Action slight and decreasing as eyes are carried to L.	Down. Action marked and increasing as eyes are carried to L.	[To L.] Action slight and decreasing as eyes are carried to L.	R. eye carried down mainly by superior oblique, L. by inferior rectus. R. eye carried toL.by internal rectus assisted by inferior rectus; L. eye carried to L. by external rectus assisted by superior oblique. Both vertical meridians rotated to R.
	Inferior Rectus	Superior Oblique	To L. action marked and increasing as eyes are carried to L.	Down. Action slight and decreasing as eyes are carried to L.	To R. Action marked and increasing as eyes are carried to L.	
	Synergists.					
	Internal Rectus	External Rectus	To L.	No action.	No action.	

An inspection of the tables just given will show that in all parallel movements of the eyes each muscle acting upon the right eye is associated with a muscle which acts upon the left eye in a precisely similar manner, and to a precisely equal extent. Such a pair of muscles, one in each eye, are termed *associated antagonists* (A Graefe).

Thus the superior rectus of one eye and the inferior oblique of the other are associated antagonists, since in all positions that the two eyes may assume, these muscles move their respective eyes to the same extent and in the same direction, so that if they acted alone they would always keep both visual lines and both vertical meridians parallel. The associated antagonists and their action may be summarized as follows:

ASSOCIATED ANTAGONISTS.

Muscle. R. eye.	Moves eye laterally to	Moves eye Vertically	Rotates upper end of vertical meridian cornea	Vertical action increasing & latent action diminishing as eyes are turned to	ASSOCIATED ANTAGONIST. L eye
External Rectus	R	No action	No action		Internal Rectus
Internal Rectus	L	No action	No action		External Rectus
Superior Rectus	L	Up	L	R	Inferior Oblique
Inferior Rectus	L	Down	R	R	Superior Oblique
Superior Oblique	R	Down	L	L	Inferior Rectus
Inferior Oblique	R	Up	R	L	Superior Rectus

The determination of the *range of excursion in associated parallel movements* comprises the solution of two distinct problems, namely, the determination of the field of binocular single vision and the determination of the field of binocular fixation. We delimit the *field of binocular fixation* by ascertaining for each direction of the gaze the point at which either one of the eyes fails to follow an object moving before the two. This can be done very conveniently with the double dot used for testing the monocular field of fixation, since the moment when either eye fails to follow the dots or when either eye fails to keep up with the other in following them, is rendered evident by a blurring of the image causing the two dots to run into one.

Hering, who used a different method (with after-images), found the binocular field to be of quite small extent, being considerably smaller than the portion common to the two monocular fields.

It seems likely, however, that his tests in this case, as in the case of the monocular field, were made upon a not altogether normal subject. My own researches, although few, to be sure, were made upon quite normal individuals. They gave the following results:*

MOVEMENTS OF BOTH EYES.

	Up.	Up and right.	Up and left.	Right.	Left.	Down.	Down and right.	Down and left.
Case I.....	38	52	45	58	52	70	80	70
Case II.....	50	52	53	56	59	70	62	70

The delimitation of the *field of binocular single vision* is effected by noting in any particular direction of the gaze the point at which one eye can no longer keep pace with the other, as evidenced by the development of an insuperable diplopia.

The field defined by joining all such points is not necessarily coincident with the field of binocular fixation, since it is quite conceivable that the two eyes following a moving object might fail to fix

*Examination made for near points.

it, but might yet both lag behind to an equal extent, so that the two images, although not formed upon the maculæ, would still be formed upon corresponding points. In this case, binocular single vision would still be present, although binocular fixation would no longer exist.

A point upon which some stress has been laid is that this method of delimiting the field of fixation gives uncertain results, since, as is alleged, many people fail to recognize diplopia in eccentric positions of the gaze. It is claimed, in other words, that the normal field of binocular single vision is quite small, and that diplopia occurs normally in looking far up, far to the right, etc., but that its existence is not suspected, because the subject under examination either fails to notice or actually suppresses one image. My own experiments, however, lead me to negative this idea completely. If we employ a candle for our test-object, and place a red glass before one eye of the individual examined, the *presence of binocular single vision* will be shown by the fact that the candle-flame appears pinkish or, more commonly yellow with a reddish border. *Manifest diplopia* will be shown by the presence of two flames, one red and the other yellow, and *diplopia with the suppression of either image* by the presence of one flame, either pure red or pure yellow. The differences presented are marked and readily appreciated by an intelligent patient when once they have been pointed out to him.

Testing in this way a large number of people with apparently normal eyes, I have uniformly found that the field of binocular single vision *extends not less than 40° in any given direction* and usually extends up to 50° or more. Indeed, most persons still get true binocular single vision, even when the eyes are carried to the extreme limit of their excursion, the field of binocular single vision being larger than either monocular field of fixation taken separately. This is but another instance of the law that the movements of the eyes, however extensive or however limited in themselves, are always under normal conditions modified in such a way as to best subserve binocular fixation and binocular single vision. Thus, as the experiments just adduced seem to show, it appears that, no matter what the maximum range of excursion of each eye separately is, the excursion of *both together*, effected by the co-ordinating action of the association-centres, is such that one eye keeps pace with the other, going neither faster nor slower, and that each stops moving when the other does. Hence, however far the object looked at may be carried in any given direction, no diplopia occurs, or, if it does, it is transient and superable.*

*These statements presuppose (1) that the visual lines are not far from parallel, i. e., the test-object should not be less than 3 feet from the eyes; and (2) the person examined should endeavor all the time to follow the object, i. e., must not look beyond it. In the latter case, of course, the test-object will seem to him double. Such diplopia is, however, usually at once superable by voluntary effort.

Each of the main associated parallel movements turning (dextroversion, or the turning of both eyes to the right, sinistroversion, or the turning of both eyes to the left, sursumversion, or parallel movement up, and deorsumversion, or parallel movement down) is apparently *presided over by a distinct nucleus (association centre)*. The precise location of these centres has not been satisfactorily determined, but the evidence of their existence from pathology is very strong, lesions in which dextroversion and sinistroversion alone are affected being not infrequent, and isolated involvement of sursumversion also having been recorded. These facts will be referred to later on.

Movements of convergence may be regarded as associated parallel movements to which a simultaneous contraction of both interni has been superadded. Thus in looking at a near object situated up and to the right there is a movement of sursumversion and dextroversion combined with a contraction of both interni, which neutralizes in part the right-hand movement of the right eye, and reinforces the right-hand movement of the left eye.

This double contraction of the interni is *presided over by a special centre (convergence centre)*, distinct from the association centres for parallel movements.

Convergence, when marked, modifies somewhat the effect of the other muscles that are acting with the interni. Thus when the gaze is directed at a near object in the median line, the superior rectus of one eye and the inferior oblique of the other no longer act as associated antagonists, the former serving mainly to adduct, and the latter to elevate the eye. In this case, in fact, the superior and inferior obliques in each eye neutralize each other completely, and the two superior and two inferior recti act as synergists to the two interni, all adducting the eye. Again, when the gaze is directed at a very near object, situated upward and to the right, the right superior rectus, since the right eye is not pointed as far to the right as the left one is, will not be working as an elevator at quite the same mechanical advantage as does the left inferior oblique. Theoretically, therefore, the right eye will lag somewhat below the left. Practically, I have not observed this to occur, although it does seem to me that the field of binocular single vision is smaller for convergent than for parallel movements.

The *maximum power of convergence* is obviously represented either by the angle formed by the two visual lines when both eyes are turned in to their utmost extent, or by the distance from the eyes of the nearest possible point upon which they can be converged. This point is called the *fusion near-point*, or, better the *near-point of convergence* (Pc.) Donders in a boy of 15 found the maximum angle of convergence to be 70°, which,

with an interocular distance of 60 mm., would signify a near-point of convergence situated 52 mm. from the centre of rotation of either eye and 42 mm. in front of the line joining the centres of both eyes, or about $1\frac{1}{2}''$ from the cornea and $\frac{3}{4}''$ in front of the bridge of the nose. Some can converge to even a greater extent. Prof. Le Conte, for example, who had acquired extraordinary facility in the use of his eyes, had a convergence-angle of nearly 90°. Schuurmann, on the contrary, found a maximum convergence-angle of only 43°, which would correspond to a convergence near-point situated about $2\frac{3}{4}''$ from the corneæ and 22″ in front of the bridge of the nose; and v. Graefe gives to the convergence-angle a value of 60° corresponding to a distance of 2″ from the eye, and $1\frac{1}{2}''$ from the bridge of the nose. My own experience leads me to regard Schuurmann's figures as expressing most nearly the results found in the general average of cases, the convergence near-point in the majority of normal persons that I have examined being situated at about 2″ in front of the nose. A distance of $1\frac{1}{2}''$-2″ may, in fact, be regarded as the normal for adults. Children often have a greater power of convergence, and in them the distance may not exceed 1″. A distance of less than 1″ denotes excessive, and one of over $2\frac{1}{2}''$ deficient convergence-power.

Another method of determining the power of convergence is by ascertaining the *strength of prism*, which can be overcome by the eyes when placed before the latter with its base out or towards the temple.

This method is analogous to that employed by Donders for determining the positive portion of the range of accommodation. The strength of prism overcome, in fact, represents the amount of *residual convergence** that the subject under examination can exercise when his eyes are adjusted for the distance of the test-object employed, just as the strength of the concave glass that he can overcome represents the amount of his *residual accommodation* under the same conditions. The amount of this residual convergence naturally varies with the distance of the test-object, decreasing as the latter is brought nearer. It also varies considerably for the same distance in different individuals, until the latter have by training learned to do what at the outset is quite difficult for them, namely, to look at a distant object and at the same time direct their eyes as if it were much nearer than it really is. When this art has been learned, it will be found that,

Normal subjects will for test-objects at a distance of twenty feet overcome prisms of 60°-70° refracting angle (equivalent to a convergence of 40°-50°), so that the maximum convergence produced in this way equals that produced in the natural fashion, i. e., by looking at a very near object.

*Often improperly called the adduction.

Convergence thus produced by prisms is at first associated with an *accommodative effort* similar to, but less than that accompanying a natural convergence of the same degree. Thus two cases that I examined showed the following amount of accommodation:

	Actual amount of convergence produced by overcoming the prism.	Accommodation exerted.	Corresponding amount of accommodation for a natural convergence of the same degree.
Case 1....	4.3°	0.25	1.25
	6.5°	0.50	2.00
	11.00°	1.50	3.25
Case 2. ..	15.5°	2.50	4.50
	19.3°	5.00	5.50

By continual practice, however, the patient may learn to relax the accommodation while maintaining the convergence, and in this way prisms of 20° or 30°, base out, may be overcome without the accommodation being used at all. I have met with an extreme instance of this sort in which the patient could, without making any accommodative effort whatever, overcome prisms representing a convergence-angle of nearly 40°.

Divergence, or the simultaneous lateral separation of the visual lines, is a process which ordinarily subserves binocular fixation, being used when the eyes fix in succession objects more and more remote. The process may, however, be performed to excess, so that the visual lines diverge from the object of fixation, as when the homonymous diplopia caused by a prism placed base in before the eye is overcome, or, on the other hand, an involuntary or voluntary crossed diplopia is produced by turning the eyes outward.

Divergence of the sort last mentioned, i. e., that giving rise to a crossed diplopia, varies greatly in amount, and, although regarded as normal by those experimenters who have acquired a peculiar facility in producing it, is probably to be classed among the abnormalities. At all events, there are not many in whom the phenomenon is habitual, or who can produce it at will, and when present, it is generally associated with lack of muscular balance, and other evidences of a pathological state.

On the other hand, a divergence produced in the act of overcoming a prism placed, base in, before the eyes, is an entirely normal phenomenon of very definite character. Its maximum amount naturally varies with the distance of the object of fixation, increasing as the latter approaches the eye. The strength, in fact, of the prism, base in, that the eyes can overcome when regarding an object at any given distance, represents the amount by which the eyes, when converged upon the object and accommodated for the latter, can diverge; just as the strength of the

convex glass that can be overcome in looking at the object represents the negative portion of the range of accommodation for the same distance. For distance, i. e., when the visual lines are parallel, the divergence* amounts quite regularly to from 3° to 5° (= divergence produced in overcoming a prism of 6° to 10°); and variations above or below these limits must be regarded as distinctly pathological.

As to the *true nature of divergence*, i. e.; whether it consists in an active muscular contraction as in the case of convergence, or whether it is simply a relaxation of the interni, allowing the eyes to return to a position of rest, there has been much difference of opinion.

Those who adopt the latter view assume that the natural position of the eyes, i. e., that in which all the muscles are fully relaxed, is one of slight divergence, parallelism itself requiring a certain tonic and constant contraction of the interni for its maintenance (Hansen Grut). Some have even thought that the position of complete relaxation is that in which each visual axis coincides with the axis of the orbit—a state of things implying a divergence of 25°-30° (Le Conte). Those who thus think, however, appear to be misled in regarding as natural a condition which is abnormal, not to say pathological. Schweigger has argued strenuously against Hansen Grut's theory and especially to his application of it as explaining the nature of divergent squint, and Schneller also has adduced a variety of arguments, which, however, are not very convincing, to prove that the function of divergence is an active process. For my own part, I believe that in the majority of cases the position of perfect physiological rest is not one of divergence and that, consequently, the lateral separation of the visual lines must be regarded as, in part at least, an active process due to simultaneous contraction of the externi. One argument in favor of this is that many people, when we test their divergence with prisms, experience a marked sense of strain analogous to that felt in overcoming prisms by converging the eyes. The latter is certainly an active process, and the former, therefore, in these cases at least, would seem to be one also. Patients, to be sure, who can diverge at will so as to produce crossed diplopia, often assure us that they do so by "relaxing" the eyes; but several observations have convinced me that this relaxation is really a muscular contraction.†

Perhaps the strongest argument in favor of the idea that divergence is a passive and not an active process is that, in the great majority of cases at least, the *diverging power, as measured by the ability to overcome prisms, base in, can not be increased at all beyond the initial amount*, shown by the subject experimented upon If, for example, in our first trial of a patient, at the maximum prism, base in, that he can overcome is one of 8°, we shall generally find

*Usually but improperly called the abduction.

†A similar instance in which an undoubted muscular contraction was described by the patient as a "relaxation" was one that I met with, in which an homonymous diplopia of 15° (prism) was produced at will. Here, of course, a condition of convergence was present, which could only have been brought about by an active contraction of the interni.

that we can not get him beyond this point by any amount of subsequent training. If divergence were a process of active muscular contraction it would seem as if it ought to be susceptible of being increased by exercise.

But whatever the nature of divergence, whether active or passive, it is certainly a distinct function of the eyes, and probably regulated by a distinct nervous mechanism. The evidence afforded by pathology, at all events, point very strongly in this direction.

Separation of the visual lines in a vertical plane (**sursumduction,** or, more properly, **sursumvergence**) is a movement which all normal eyes can perform. It is, however, very limited in amount, not normally exceeding 1° or 1½° (= the divergence produced by a prism of 2° or 3°). It is evidently an active process associated with a sense of considerable strain, and appears susceptible of being increased by exercise, particularly in those that have a natural or acquired vertical deviation (hyperphoria).

The power of producing **convergence or divergence of the vertical meridians** of the two eyes, the visual lines remaining parallel, is a subject about which very little is known. Even the experiments of Helmholtz, which seem to prove its existence, have been called into question by some, although probably without sufficient reason.

APPENDIX. The analysis of the complicated problems involved in the study of the movements of the eyes may be facilitated by reference to the diagram (Fig. 2), which represents the field of fixation of a normal eye having a rather extensive power of motion.

PROJECTION OF THE FIELD OF FIXATION AND OF THE FIELD OF ACTION OF EACH OF THE OCULAR MUSCLES.

C, projection of extremity of line of sight (point of fixation), when eye is in primary position; D, E, O, projection of same when eye is abducted 18°, 30°, and 50°, respectively; B, A, P, projection when eye is adducted 20°, 50°, and 60°, respectively. The distance CO. represents the maximum degree of excursion of the eye outward. Most of this movement is effective by the *external rectus*, but a certain portion especially towards the outer end of the excursion is accomplished by the united action of the two obliques (see *infra*). The distance CP. represents the maximum range of excursion of the eye inward. This inward movement is effected mainly by the *internal rectus*, assisted especially towards the end of the excursion by the superior and inferior recti (see *infra*).

The black lines AA_1, BB_1, CC_1, DD_1, EE_1, represent the amount and direction of the movement produced by the *superior rectus* when the eye is respectively adducted 50° (A); adducted 20° (B); in the primary position (C); abducted 18° (D); and abducted 30° (E). The red lines AA_2, BB_2, CC_2, DD_2, EE_2, represent the amount and direction of the movement effected by the *inferior oblique*, and the dotted lines AA_3, BB_3, CC_3, DD_3, EE_3, the movement effected by the in-

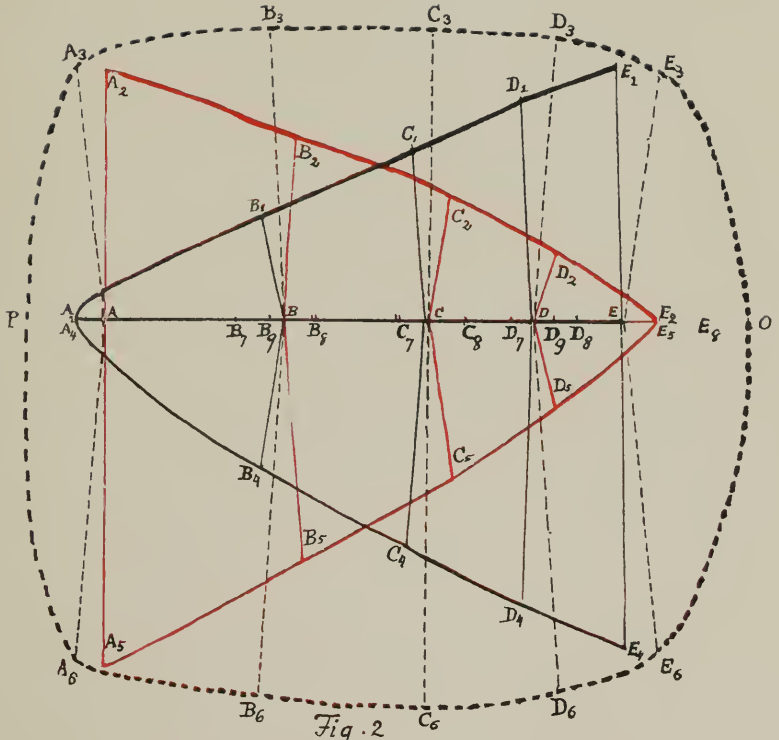
P
O
A3
B3
C3
D3
E3
A2
B2
C2
D2
E2
A1
B1
C1
D1
E1
A
B
C
D
E
A4
E4
E5
A5
B5
C5
D5
A6
B6
C6
D6
E6

Fig. 2

ferior oblique and the superior rectus acting together. Similarly, the black lines AA_4, BB_4, CC_4, DD_4, EE_4 represent the lines of action of the *inferior rectus*; AA_5, BB_5, CC_5, DD_5, EE_5, those of the *superior oblique*; and AA_6, BB_6, CC_6, DD_6, EE_6, those of the two latter muscles combined. The heavy black lines $A_1B_1C_1D_1E_1$ and $A_4B_4C_4D_4E_4$ represent the limits of the *fields of action* of the superior and the inferior recti; and the heavy red lines, $A_2B_2C_2D_2E_2$ and $A_5B_5C_5D_5E_5$ the limits of the fields of action of the inferior and the superior obliques. The heavy dotted line $A_6PA_3B_3C_3$, etc., represents the limit of the *field of fixation*.

It will be seen from the diagram how the *vertical* (elevating and depressing) action of the superior and inferior recti increases, and how the vertical action of the obliques decreases progressively as the eye is carried from a position of marked adduction (A) to one of moderate abduction (E). It will also be apparent how the *lateral* action of each of these muscles diminishes as its vertical action increases; so that the adductive power of the superior and inferior recti shows a progressive diminution, and the abductive power of the two obliques a progressive increase as the eye passes from A to E. That is, at A (i. e., when the eye is adducted 50°) the superior and inferior recti have no vertical action at all, but simply adduct the eye through a comparatively large extent, while the two obliques have no lateral action at all, but simply elevate and depress the eye.

At E, on the other hand (when the eye is abducted 30°), the two recti no longer act as adductors at all, but simply elevate and depress the eye; and the two obliques no longer exert any vertical effect, but combine to carry the eye outwards, their abductive action, in fact, being here at its maximum.

At A the *superior rectus and the inferior oblique acting together* to their full extent will carry the eye up and noticeably inwards to A_3); since here the adductive action of the rectus is at its maximum and besides is not balanced by any opposing abductive action on the part of the oblique. Similarly at E the two muscles acting together will carry the eye upwards and noticeably outwards. In intermediate positions, as at B and D, the lateral action of one muscle will partially counteract that of the other, so that the net lateral effect will be less. E. g., at B the adductive action of the superior rectus is less than it was at A and moreover is now opposed by a moderate abductive action on the part of the inferior oblique, so that the net adductive effect is but slight. The eye, therefore, here is carried up by the inferior oblique, assisted somewhat by the superior rectus, and is also carried slightly inwards (to B_3). At D, on the contrary, it is carried upwards mainly by the superior rectus, assisted somewhat by the inferior oblique, and is also carried slightly outwards (to D_3), by the preponderating lateral (abductive) action of the latter muscle. At C (the primary position) the adductive action of the superior rectus apparently balances the abductive action of the inferior oblique, and hence the effect of the two elevators acting together will be to carry the eye straight upwards.

In a similar way the *inferior rectus and the superior oblique acting together* will carry the eye down and in (AA_6, BB_6), down and out (DD_6, EE_6), or straight down (CC_6), according as the eye is already adducted, abducted, or in the primary position.

The *superior or inferior rectus acting together* with just sufficient force to neutralize each other's vertical action, will combine to adduct the eye, the adducent effect being forcible at A ($=AA_1+AA_4$ or AA_7), and diminishing gradually to E where it is zero. At E, therefore, i. e., when the eye is abducted 30°, the inferior and superior

recti acting together will produce no movement and hence in this position are direct antagonists.

Similarly the *superior and inferior obliques acting together* neutralizes each others' vertical action, but combine to abduct the eye. The abducent effect is greatest at E ($= EE_2+EE_5=EE_8$), and diminishes progressively to A, where it is zero. In the latter position, therefore, i. e., when the eye is abducted 50°, the two obliques, acting simply to elevate and depress the eye respectively, are direct antagonists.

The four muscles, *superior and inferior recti and superior and inferior obliques acting together* with the force required to neutralize each other's vertical action, will produce a lateral effect varying with the amount by which the eye is already abducted or adducted. Thus if the eye is already adducted 50° (to A), a position in which the obliques exert no lateral action at all, the total effect of the four muscles will be to carry the eye quite a little distance further inward (to A_7). If the eye is adducted only 20° (B), the resultant action of the four muscles will be the difference between the adductive action of the two recti (BB_7), and the less marked abducent action of the two obliques (BB_8); i. e., the eye will be adducted slightly (to B_9). At C (the primary position), the abducent action of the obliques balances the adducent action of the superior and inferior recti, so that the contraction of the four muscles will cause the eye to remain stationary. At D, on the contrary, the abductive effect will preponderate somewhat, and the eye, already abducted 18°, will be carried still further moderately outwards (to D_9). At E, i. e., when the eye is abducted 30°, the abducent effect is still more pronounced ($= EE_8$).

It is thus apparent that if all four muscles act together they will, if the eye is being adducted or abducted, *tend to carry it still further in the direction in which it is going*. They will, therefore, reinforce the external rectus in abducting and the internal rectus in adducting the eye, and the amount of the reinforcement will increase in proportion as the eye is already abducted or adducted. So that the internal rectus, for example, when it begins to contract (i. e., is just leaving the primary position at C) will receive little or no aid from the contraction of the other four muscles, but, as it continues to act, (e. g. at B), will be more and more assisted by them, and finally when it reaches the limit of its contraction (at A) and is consequently working at a great mechanical disadvantage, will be strongly reinforced. In like manner the external rectus will, as its own efficiency diminishes with the increasing abduction of the eye, be assisted more and more by the simultaneous contraction of the other muscles. And it is altogether probable that it is in this way that the ontward and inward excursion movements of the eye are rendered regnlar and uniform.*

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*It may be noted that the action of the superior and inferior recti in compensating for an increasing feebleness of the internal rectus is shown in another way also. The internal rectus, as Weiss has pointed out (Arch. f. Augenheilk; Vol. xxix), acts very much more feebly when the divergence of the orbits is great, i. e., when the orbits are shallow and the eyes far apart. But it is under just these conditions that the superior and inferior recti act to most advantage as adductors, since, the greater the divergence of the orbits, the greater the angle which the line of action of these two muscles makes with the antero-posterior axis of the eye, and the greater consequently is the lateral effect which they are able to exert.

Our diagram may also be used to illustrate the action of the *associated antagonists*. If alongside of Fig. 3, which represents the field of fixation of the right eye, we place one representing the field of fixation of the left eye (which may be done by turning Fig. 3 end for end, so that O is on the left and P on the right of the figure), we shall see how the field of action ($A_2B_2C_2D_2E_2$), of the inferior oblique of the right eye agrees in all respects with the field of action of the superior rectus of the left eye. So also of the other associated antagonists (R. superior rectus and L. inferior oblique; R. inferior rectus and L. superior oblique; R. superior oblique and L. inferior rectus).

Another point elucidated by the diagram is the amount and kind of *torsion movement* (rotation of vertical meridian of the cornea) produced by the various muscles. That is the line BB_1 represents the fact that when the eye is adducted to B the superior rectus will not only carry the eye itself upwards and inwards (to B_1), but will also rotate the vertical meridian of the cornea so that the latter will have the inclination BB_1, i. e., will be inclined inwards. In a similar way, the inferior oblique in the same situation will not only carry the eye upwards and outwards (to B_2), but will also rotate the vertical meridian of the cornea outwards, so that it will have the direction BB_2. And the combined action of the two muscles will be to give the vertical meridian the inclination BB_3, i. e., one of slight rotation inwards. So also BB_6 represents the inclination of the vertical meridian of the cornea (viz., with the upper end rotated inwards), when the eye is carried downwards from a position of adduction (B). Again, the fact that CC_3 is strictly vertical shows that when the eye is in the primary position it is not only carried straight upwards by the combined action of the two elevators, but its vertical meridian also remains vertical during the ascent.

It will also be observed that the combined action of the superior and inferior recti or of the superior and inferior obliques, or of all four muscles together will be not only to keep the eye in the horizontal plane (in the line OP), but also to keep its vertical meridian from rotating either to the right or to the left, as the eye is carried outwards or inwards.*

In fact, all the various applications of the laws of Donders and Listing may be deduced from the study of this diagram.

Finally the diagram shows the *limitation of the field of fixation and the kind and amount of diplopia present in paralysis* of any one of the ocular muscles. Suppose, for example, that the superior rectus is paralyzed. Then the field of fixation while normal below, will, since the inferior oblique is the only elevator left, be represented above not by $A_3B_3C_3D_3E_3$, but by $A_2B_2C_2D_2E_2$. In other words, when the attempt is made to elevate the eye as far as possible, it will stand at A_2, instead of A_3, at B_2, instead of B_3, etc. Since its fellow eye has a normal field of fixation and hence under the same conditions rises to A_3, B_3, etc., the difference in position of the two eyes and consequently also the amount and kind of diplopia produced will be represented by the difference between A_2 and A_3, B_2 and B_3, etc. The diagram thus gives us a graphic representation of the fact that in paralysis of the

*E. g., at C the inward rotation of the vertical meridian produced by the superior rectus will be represented by the angle C_3CC_2; this inward rotation will be neutralized by the equal outward rotation C_6CC_4, produced by the inferior rectus: and hence the combined effect of these two muscles will be to keep the vertical meridian from rotating either one way or the other.

superior rectus the vertical diplopia increases rapidly when the eye is carried upwards and outwards, while the lateral (crossed) diplopia increases as the eyes are carried upwards and inwards.

The diagram may also be utilized to map out the field of fixation in cases of *combined paralyses*. Thus the field of fixation in a case of paralysis of both the superior rectus and the superior oblique would be represented by $A_2B_2C_2D_2E_2E_4D_4C_4B_4A_4$; and in a combined paralysis of the superior and inferior rectus by $A_2B_2C_2D_2E_2D_5C_5B_5A_5$.

III.

THE TESTS EMPLOYED AND THEIR SIGNIFICANCE.

The *object* of the various tests that we make use of is to determine the following data:

(1). The precision and steadiness with which binocular fixation is effected (Static Tests).

(2). The ability of the eyes to move in various directions while still maintaining binocular fixation (Dynamic Association-tests).

(3). The ability of the eyes voluntarily to deviate from the position of binocular fixation (Dynamic Disassociation-tests).

These tests may be performed both for distance (with the visual lines parallel) and for near (with the visual lines converged).

The chief tests for *binocular fixation* are:

(1). **Inspection with both eyes uncovered.** This gives us an approximate idea as to whether both eyes are directed at the same object, a non-fixing eye appearing to deviate in, out, up, or down, according to circumstances.

In making this test we must be careful not to be misled by the presence of a large angle alpha, which may simulate a deviation where none exists. Any error on this score will be prevented by comparing the findings with those of the screen test; for a deviation, great enough to be noticeable upon simple inspection, will certainly give evidence of its presence by a distinct movement of the eyes when the cover is shifted from one eye to the other.

(2) **Fixation and Diplopia Tests.** A patient with normal eyes and perfect binocular fixation, will see distinctly with either eye alone, or with both together, and will also see single. If either eye or both fail to fix the object looked at, that object will appear blurred *(Fixation-test)*, and if one eye fixes and the other does not, the patient will in general see double *(Diplopia-test)*, the image of the fixing eye being clear and that of the other more or less shadowy and indistinct. The kind of diplopia present indicates the nature of the deviation. Thus an *homonymous diplopia* (i. e., one in which the image formed by the right eye is on the right side, and that formed by the left eye on

the left side) signifies abnormal convergence of the visual lines; a *crossed or heteronymous diplopia* (in which the image of the right eye is on the left side and vice versa) signifies lateral divergence; and *vertical diplopia* (in which one image is higher than the other) signifies vertical separation of the visual lines, so that one is higher than the other. The last-named variety may be further differentiated into *right diplopia*, in which the image formed by the right eye is below (indicating the condition in which the right visual line is the higher), and *left diplopia*, in which the contrary conditions prevail.

The *amount* of diplopia is precisely proportional to the amount of deviation. It may be measured either by estimating the linear distance between the two images, the distance of the object looked at being also known,* or by determining the strength of the prism which appropriately placed, will correct the diplopia.†

In order to differentiate the double images it is convenient to use a light as a test-object, and have a red glass placed before one of the eyes. By thus giving the two images a different color‡ we enable the patient the better to distinguish between the two and recognize the fact that diplopia exists; and, moreover, since the red flame must belong to the eye covered with the red glass, we can determine from the patient's statements as to the relative place of the red and white images, whether we are dealing with homonymous or crossed (lateral) or with right or left (vertical) diplopia.

(3) **Equilibrium Test.** This is simply a variety of the diplopia test. It consists of two steps. In the first an artificial homonymous diplopia is produced by means of a prism of 12° or more, placed base in, before the eyes. If the two images thus produced are on a level, the visual lines themselves are on a level. If, however, the right-hand image should be lower, there is really a natural right diplopia present in addition to the artificial homonymous diplopia, i. e., the right visual line is higher, or, to use Stevens' nomenclature, there is right *hyperphoria*. The amount

*A linear distance of 1″ between the images is equivalent to a deviation of 1½° in the visual lines when the test-object is 1 metre distant and to ¼° when the latter is 20 feet distant.

†A prism rarely measures the full amount of the diplopia, as a prism which slightly undercorrects the latter nevertheless brings the double images so close together that the residual correction can be and is effected by the eyes themselves.

‡A similar difference in character may be imparted to the images by placing a Maddox rod or a Stevens' sphere before one eye; but the red glass is simpler and, in comparison with the Maddox rod at least, is less confusing to the patient and less apt to give ambiguous results.

of this latter may be measured by the degree of prism, which, placed base down before the right eye, will rectify the diplopia, i. e., will bring the images on a level. In the next step of the test, a strong prism is placed base down before the right eye, producing a marked vertical (left) diplopia. If both eyes are properly adjusted for the object of fixation, the two images will be in a vertical line. If, however, the upper image is to the right of the lower, there is really, besides the artificial vertical displacement, a natural homonymous diplopia, or, to use Stevens' expression, there is an *esophoria*. Similarly, if the upper image is to the left of the lower, there is really a crossed diplopia or *exophoria*. In either case the amount of the esophoria or exophoria may be measured by the strength of the prism which, placed base out or base in will rectify the diplopia, i. e., will bring the two images into a vertical line.

In Stevens' phorometer, which is the best instrument for this purpose, the measurement of the deviation is effected, not by placing additional prisms before the eyes, but by revolving the prism that has been used to produce the initial lateral or vertical diplopia until the images are truly horizontal or vertical. The amount of rotation is read off on an arc graduated so as to indicate directly the amount of hyperphoria, esophoria, or exophoria present.*

One defect of the equilibrium test is that patients often try involuntarily to bring the two images into line and thus appear to have no deviation of the visual lines, although one actually exists. On the other hand, the involuntary movements set up in the attempt made to compare two similar images placed at a distance from each other may cause a deviation to be simulated where none is present. I have seen this occur not infrequently—sometimes to a very marked degree. The equilibrium test being thus apt to set up a certain amount of muscular tension and hence disturb the true relation of the visual lines, is in actual practice best performed after the tests next to be described in which the eyes are under more normal conditions.

(4) **Screen Test.** This depends upon the fact that the tendency to binocular fixation is so strong that it still persists, even when one eye no longer sees the object of fixation. If, therefore, a card be placed before the left eye, and the gaze be directed at a distant object, the left eye will, in case there is no disturbance of innervation causing it to deviate, look straight at the object, just as if the latter were still visible. If now the card is shifted from the left eye to the right, the former being already properly directed, will not have to change position in order to fix the ob-

*The same thing may readily be done with the ordinary trial-frame, if we use in it a 12° prism and recollect that with this each rotation of 5° from the horizontal represents 1° of hyperphoria, and each rotation of 5° from the vertical 1° of esophoria or exophoria.

ject, and will hence remain stationary. If, however, the left eye when screened, deviates in any way, e. g. outward, it will, when the screen is transferred to the right eye, have to turn inward, or to the right, in order to fix the object, and the amount of its excursion inward *(movement of redress)* will be precisely equal to the amount of its previous deviation. At the same time that the left eye turns inward, or to the right, in order to perform fixation, the right eye, which is now covered by the card and which, according to the law of associated parallel movements, receives an impulse to move to the right equal to that communicated to the left eye, will move outward.

Whether it moves outward *to the same extent* that the left eye moves in or not, depends upon the relative ability of the muscles of the two eyes to respond to the stimulus imparted to them. If, for instance, the left internus is weak (paretic) a very strong impulse will be required in order to make the muscle contract enough to cause the eye to move in to the proper extent. According to the law of association, an equally powerful impulse will be communicated at the same time to the right externus; and, if the latter is normally strong, it will respond much more efficiently to this impulse than did the weak internus of the other eye, and will, consequently, carry the right eye out much further than the left eye was carried in.

It may be stated as a general rule (to which, however, there are not a few exceptions) that in concomitant deviations the deflection behind the screen, and hence also the movement of redress that the eye makes when the screen is removed, are equal for the two eyes, and that in non-concomitant deviations they are unequal, being greater in the eye which has the more powerfully acting muscles.

The screen test may also be used to ascertain *which of th two eyes habitually fixes.*

In doing this the screen instead of being shifted from one eye to the other is simply removed from the eye before which it is placed, leaving both eyes uncovered. Each eye under these circumstances will deviate when the screen is in front of it, and the other eye will fix. If now the eye that is behind the screen is the one that in binocular vision is regularly employed for fixation, it will move into the position of fixation as soon as the screen is taken away, and the other eye will deviate. If, however, the eye that is behind the screen does not ordinarily perform fixation, it will not move when unscreened, and the other eye will continue to fix, i. e., will remain steady in its place. That is, the fact that the eyes perform a movement of redress when the right is unscreened and both are left open indicates that the right eye habitually fixes. If no movement takes place, when the right eye is unscreened, the latter can not be the eye that habitually fixes; and if no movement takes place when the right eye and the left alternately are unscreened, there must be an alternating deviation, i. e., one in which either eye indifferently is used to fix with.

The *amount* of deviation behind the screen, or of the movement of redress made by the eye from which the screen has been removed, may be roughly estimated by marks made upon the lids or may be more accurately determined with the perimeter or by some of the various strabometric methods which have been well described by Maddox (*Archives of Ophth.*, XXI., 1, 1892). An angular deviation of 1°-2° is generally sufficient to produce a noticeable deviation behind the screen.

Finally, it must be noted that the screen test is *valueless unless the patient can be got to fix* with the uncovered eye. Hence, the test is of no service in those who, owing to a deviation of long standing, have lost the power of fixation; and it may likewise prove nugatory in children who fail to keep their gaze directed at the object that they are told to look at.

(5) **Parallax Test.** When the screen test is employed, the patient, if his eye deviates behind the screen, will in general notice a movement of the object whenever the screen is shifted. This movement is called the *parallax*, and, if the test-object is so placed as not to be projected upon any surface back of it (e. g., if it is a spot upon a blank wall), furnishes a valuable indication of the amount and character of the deviation. The perception of this movement is really nothing but the perception of a diplopia, which differs from ordinary diplopia in the fact that the *two images are seen in succession, instead of at the same time*, and, as they occupy different places, give the impression of a single image which has moved from one place to another. Thus, if there is convergence (esophoria), the right eye, when unscreened, and before it has had a chance to assume the position of fixation, sees the object a little further to the right than the left eye saw it, i. e., the object appears to have moved from left to right *(homonymous parallax)*. If, on the other hand, there is divergence (exophoria), the right eye will when unscreened see the object a little further to the left than the left eye did when it was fixing, i. e., the object appears to have moved from right to left *(crossed parallax)*. So, too, right hyperphoria is indicated by the fact that the object appears to move down when the right eye is uncovered *(right parallax)*, while in left hyperphoria the object seems to move up *(left parallax)*. These various movements are noticeable even when the deviation is very slight; a hyperphoria of 0.1°, for example, being made appreciable by a distinct up and down movement of the object.

The *amount* of the parallax may be measured by the strength of the prism which, placed before the eyes, will neutralize the

movement. Homonymous parallax will be neutralized by a prism base out, a vertical parallax by a prism with the base up or down, etc. The fact of neutralization or reversal is generally indicated by the patient with great precision.

Tests for the Associated Parallel Movements. The ability of the eyes to perform associated parallel movements, i. e., *the range through which they can move in any direction and still carry on binocular fixation*, is tested in the same way as the ability to maintain binocular fixation while in the primary position. All the tests just described are applicable. Thus inspection enables us to say whether the movement of the eyes in, out, up, or down, is too slight or too excessive; also the point where one eye ceases to keep up with the other, this being shown by the fact that the former visibly lags behind or wavers in its course. In this way we may map out the *monocular* or *binocular field of fixation*, as may also be done more accurately by the fixation test (with the double dot, as already described). So, too, by the diplopia test we map out the *field of binocular single vision*, and thus also determine whether the eyes follow each other to a normal extent or not. With the same object in view we apply the screen, parallax, and equilibrium tests to ascertain if there is any visible deflection, parallactic movement, or heterophoria within the limits of the normal field of fixation, and, if so, where they begin, and in what direction they increase.

By these various means we determine *whether the movements in any given direction are excessive or restricted*. In this regard, inspection and the diplopia and screen tests are practically the most applicable. The mapping out of the field of fixation is laborious and, for the reasons already given, the results obtained are very uncertain, unless a series of examinations upon the same patient happen to be quite concordant.

On the other hand, the diplopia test is readily applicable, and in my experience gives much more constant and reliable results. Diplopia may, to be sure, occur normally, as a transient phenomenon *(physiological diplopia)* in most people when the gaze is carried far towards the periphery of the field of fixation; but such diplopia, as already stated, is inconstant and superable by voluntary effort. A diplopia occurring under all circumstances as soon as the gaze has been carried from 35° to 40° from the primary position, in any given direction, indicates an abnormal weakness. This again may be temporary, and a diplopia of this sort occurring about equally far in all directions from the periphery *(concentric contraction of the field of single vision)*

indicates a temporary enfeeblement of all the ocular muscles such as may happen in neurasthenia and form one of the evidences of a general depression of the muscular forces. On the other hand, a diplopia, insuperable by voluntary effort and constantly occurring as soon as the gaze is carried 30° or less in any given direction from the primary position, indicates a true weakness or paresis, of some one of the ocular muscles *(paretic diplopia)*. The differential diagnosis of this condition, based upon the character of the diplopia, will be touched upon later.

Tests for Convergence. The *tests for binocular fixation* in convergence are the same as those for distance, namely, inspection, the fixation and diplopia tests, the screen and parallax tests, and the equilibrium test. All of these, in fact, are habitually applied with the test-object held at the ordinary reading distance, as well as at a distance of 20 feet. The test-object itself for the examination at near points should, as Randall has well said, be something requiring accurate fixation (e. g. a pen-point or fine dot, instead of the finger which is habitually used). In determining the parallax, some device such as a dot on a large card, which does not allow the test-object to be projected upon any surface beyond it, should be employed.

It must be borne in mind that, while orthophoria—absence of deviation—is the ideal state for distance, *a slight amount of divergence is physiological for near*. Thus, in testing at 12″, we expect to find with the phorometer an exophoria of 3° to 6°, and with the screen a crossed parallax of the same, or a somewhat less amount; and orthophoria at this range is actually to be regarded with suspicion, as probably indicating an undue tendency to convergence. This fact does not militate against the existence in these cases of true binocular fixation for reading, or other occupations requiring precise adjustments.

The *tests for the associated movements in convergence*, likewise are made in the same way as for the associated movements at a distance. In making the diplopia test at near it is best, if using the candle, to hold the latter not less than 30″ from the eyes, so as to reduce as much as possible the effects of projection. If it is desired to determine the field of binocular single vision for closer ranges, the effects of projection may be obviated by using for a test-object a dot on a large card, the latter being tilted so as always to be perpendicular to the patient's line of sight.

A further important fact to determine in testing movements of convergence is the *convergence near-point* (Pc). This is ascertained by carrying a fine object nearer and nearer to the eyes, un-

til the latter can no longer be converged upon it, or until it appears double. The distance of the object from the root of the nose may then be measured. Notice at the same time should be taken as to which eye is the first to deviate when the limit of convergence is reached. The same test should be repeated from either side, the object of fixation being first placed at some point A to the right of the middle line and then carried directly towards the left eye L. The latter obviously will not have to change its position of adduction, but the right eye, R, in order to follow the object, must swing inwards through a considerable arc, A C P. If, on repeating the test with the left eye, one of the two is found

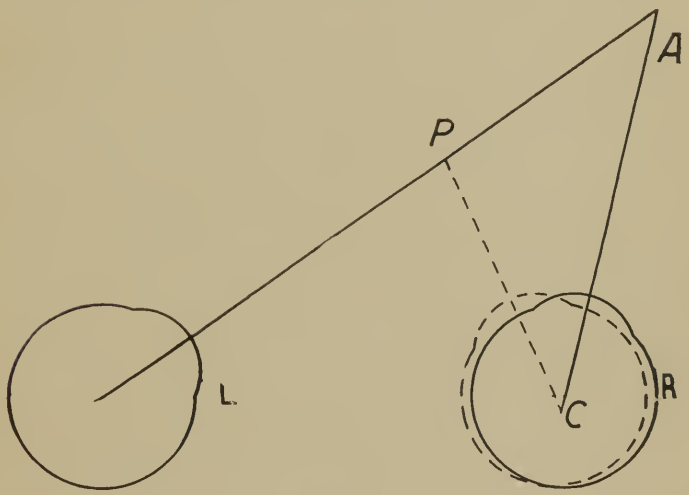

Fig. 3.

to sag off from the test-object much sooner than the other eye does, the former must have a relatively weak adducting power.

Not only the distance of the convergence near-point, but also the *ability of the eyes to maintain convergence* at this distance, should be noted.

Lastly the convergence must be tested by determining the maximum *strength of prism placed base out* before the eyes, which the latter can overcome when looking at a distant object. Usually, if the patient can at the outset overcome a prism of 20° refracting angle, with ease, we assume that he could readily learn to do two or three times as much if sufficiently exercised, and we consider his prism-convergence as normal. Exceptionally, especially in cases of convergence-insufficiency, we find that the

prism-convergence even after repeated trials cannot be got above 10° or 12° (prism), and that even this amount is hard for the patient to do and still harder to maintain.

As the exercise of the prism-convergence not infrequently begets a condition of convergence-spasm, it is generally best to defer testing the convergence in this way until after the divergence has been determined.

It is well in testing the convergence by means of adducting prism to *ascertain how much accommodation* the patient is associating with it. This can be done by using the test-types for the object of fixation and finding what concave glass is required to give the patient full sight. It will generally be found that by repeated practice with an object of this sort, the strength of the concave glass can be gradually diminished—i. e., the patient gradually acquires the ability, when looking at a distant object, to converge without using his accommodation. A case in which extreme facility in this respect was acquired has already been spoken of. It is often important therapeutically to effect a dissociation of this sort between accommodation and convergence, especially in cases of convergence-insufficiency.

Tests for Divergence. The diverging power is determined by the amount of prism placed, base in, before the eyes, which the latter can overcome when looking at a distant object. The strength of prism thus overcome varies in normal cases from 6° to 8° (refracting angle). A divergence of less than 5° (prism) means insufficiency, and one of over 9° an excess of diverging action.

Tests for Sursumvergence. The sursumvergence, i. e., the amount by which the eyes can diverge in a vertical plane, is determined by the strength of prism placed base up or down before the eyes, which the latter can overcome when looking at a distant object. The *right sursumvergence* (in which the prisms are so adjusted as to cause the right visual line to be the higher of the two) and the *left sursumvergence* should both be ascertained. It is usually best to leave some interval of time between the two tests, as after making the effort required to produce right sursumvergence (or left deorsumvergence) it is difficult at once to perform the contrary action.

A difference of 1° or more between the right and left sursumvergence or, in any case, a sursumvergence exceeding 3° (prism), indicates the probable existence of a hyperphoria.

Way in which the Tests are applied in Practice. In practice I have found it best to apply the tests in the following order:

(1) *Inspection.* I note the apparent relations of the eyes in the primary position and also for associated parallel and convergent movements, using for the purpose some rather fine test-object such as a pen-point which the patient is made to follow with the eyes as it is carried in different directions. Any very obvious deflection, e. g., a marked concomitant strabismus or a paralytic squint, can be made out at once by this means alone.

(2) *Screen and Parallax Tests.* These are made simultaneously. First, a test-object 20 feet off is taken, and then one at the ordinary reading distance. If inspection has revealed any marked deviation or one which increases notably in any given direction of the gaze, the screen test also is applied in different portions of the field of fixation in order to corroborate these findings.

(3) *Equilibrium Test* both for distance and near with the phorometer or with prisms in the trial-frame.

(4) *Test for Divergence* by means of prisms placed base in before the eyes (Abduction-test of most authors).

(5) *Determination of the Convergence Near-point* both in the median line (test for bilateral convergence) and also, as has been previously explained in the course of this brochure, in lateral positions of the gaze (test of eccentric convergence).

(6) *Test for Convergence* by prisms placed base out before the eyes (Adduction-test of most authors).

(7) *Diplopia Test* with candle at 40″ or more, and sometimes also with card and dot at 12″. In order to make this test available for diagnosis we must have some ready method of recording which shall indicate, not only the character of the diplopia, but also its approximate amount, the point at which it begins to appear, and the way in which it increases or decreases in different directions of the gaze.*

These comprise all the tests really necessary, and all these can in most cases be readily performed within ten minutes. If further tests are thought requisite, the *Sursumvergence Test* (8)

*A sample of the scheme which I have adopted for my entries is as follows:

Eu 20°, Er 25°—DL; Eu 25°, Er 30°—DL 2°, DX 2°, which would mean that when the eyes were carried 25°, to the right and 20° up from the primary position vertical diplopia appeared; the image of the left eye being lower (left diplopia); and that when the eyes were carried 30° to the right and 25° up the image of the left eye was 2° below and 2° to the right of that found by the right eye (i. e., there was a left and crossed diplopia of 2° each).

and the mapping out of the Field of Fixation (9), may be undertaken. If they are, they should be left to the last, as they generally cause considerable strain of the eyes, and hence, if performed early, are apt to derange the normal relations of the eyes, and thus interfere with any tests that may be made afterwards.

If the patient is ametropic or presbyopic, the various tests enumerated should be made both with and without the correcting glasses, in order to ascertain the effect of the latter upon the muscular condition; and other factors that might modify the latter, e. g., the existence of atropine mydriasis, should also be noted.

(To be continued.)

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A NEW CLASSIFICATION OF THE MOTOR ANOMALIES OF THE EYE, BASED UPON PHYSIOLOGICAL PRINCIPLES.

THE PRIZE ESSAY OF THE ALUMNI ASSOCIATION OF THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK, FOR 1896.

By Alexander Duane, M. D.,

NEW YORK.

Part 2. Pathology.

IV.. Classification of Ocular Deviations. Nature of functions that may be affected.—Nature of lesions affecting these functions.—Hypokinesis, Hyperkinesis, Parakinesis.—Variation in degree of the lesions. Superable and insuperable deviations (Heterophoria and Squint).—Classification propounded.—Comitant and non-comitant deviations.—General diagnostic laws based upon the presence or absence of comitancy.—Transformation of non-comitant into comitant deviations.

V. Anomalies of the Individual Muscles. Muscular under-action and over-action. Three main varieties.—Structural muscular deviations. Structural (Muscular) squint (Schneller's cases). Structural heterophoria.—Insertional squint and heterophoria.—Innervational deviations. Muscular Paresis. Muscular Spasm; varieties and cases.—Symptoms and Differential Diagnosis of the different Varieties of Muscular Over-action and Under-action. Identity in the symptoms presented by the three varieties. Congenital deviations. Course of acquired deviations. Diagnosis between under-action and over-action. Slight weakness and over-action (explaining cases of heterophoria and particularly hyperphoria). Diagnosis by the double images. Principles and diagnostic tables. Deductions from the tables. Treatment of muscular over-action and under-action.—Tremor of individual ocular muscles; Unilateral nystagmus.

VI. Anomalies of Associated Parallel Movements. Hypokinesis. Paresis and insufficiency of associated parallel movements. Spasm of associated parallel movements. Peculiar case of spasm observed by the author.—Parakinesis of associated movements (Nystagmus). Theory of nystagmus.

VII. Anomalies of Convergence. Hypokinesis. Paralysis of convergence.—Convergence-insufficiency. Signs. Nature and etiol-

ogy. Varieties of non-accommodative convergence-insufficiency. Accommodative convergence-insufficiency; varieties. Course. Complications. Symptoms. Treatment.—Hyperkinesis. Spasm of convergence. Convergence-excess. Signs. Etiology. Varieties of non-accommodative and of accommodative convergence-excess. Course. Complications. Symptoms. Treatment.

VIII. Anomalies of Divergence. Hypokinesis.—Divergence-insufficiency. Signs. Etiology. Idiopathic and secondary divergence-insufficiency. Differentiation of the two forms. Course. Symptoms. Treatment. — Hyperkinesis. Divergence-excess. Signs. Etiology. Primary and secondary divergence-excess. Differentiation of the two. Course. Symptoms. Treatment.

IX. Anomalies of Sursumvergence. Hypokinesis. Sumsurvergence-insufficiency. — Hyperkinesis. Sursumvergence-excess. Sursumvergence-hyperphoria and vertical strabismus. Peculiar cases; anatropia and catatropia.

X. Anomalies of Rotation Movements.

XI. Recapitulation. Nature of Outward, Inward and Vertical Deviations (Exophoria, Esophoria and Hyperphoria). Varieties of each and their differentiation.

IV.

THE CLASSIFICATION OF OCULAR DEVIATIONS.

We have seen in Section II. of this brochure that as physiologists we have to consider not only the movements of the individual muscles and the relative power of the latter, but also, and more particularly, the associated movements of the eyes produced by the coördinated actions of these muscles. It seems obvious that the same principles which we apply to the physiology of the ocular movements should also be applied to their pathology, and that in classifying motor disorders of the eye we should concern ourselves *not simply with the disorders and weaknesses of the muscles as such, but also with the affections of all the various movements of which either eye alone or both eyes together are susceptible.* Reasoning in this way, we shall ask if we do not in actual practice meet with pathological conditions which may be classified into the following groups, corresponding to the physiological groups which we have already studied?

(1) Disorders of individual muscles.
(2) Disorders of associated parallel movements.
(3) Disorders of convergence.
(4) Disorders of divergence.
(5) Disorders of sursumvergence.
(6) Disorders of the rotation (or swivel) movements.

I think that we have a sufficient number of clinical facts to enable us to give an affirmative answer to this query.

For example, we meet with quite a number of cases in which the power of convergence is weak—a state that we might attribute (as indeed it often is attributed) to an intrinsic weakness of the interni, were it not for the fact that the working of the latter in associated parallel movements is quite normal. Here then, evidently, it is the function of convergence that is at fault and not the action of the interni per se. Similarly we find excessive divergence action which can not be attributed to weakness of the interni, since the latter act normally both in convergence and in lateral movements, nor yet to excessive strength of the externi as such, since the latter in associated parallel movements do not carry the eye too far outward. In this case, therefore, it is the function of divergence, and not the power of the interni, or externi, that is at fault. Both this condition and the preceding one exhibit the common symptom of divergence in fixation and would hence ordinarily be classed together as examples of exophoria; but in classifying them thus we should be naming not the disease but the symptom. The symptom, moreover, while the most obvious, is not necessarily the most important feature of the condition in question; and in our therapeusis we aim not so much to abrogate the exophoria per se, as to remove the state (defective convergence, excessive divergence) causing the exophoria.

Similar instances might be given of other varieties of motor disorders in which also *the function involved is an ocular movement and not an ocular muscle.* The consideration of these, however, will be deferred until later when they can be discussed more in detail; those given above being regarded as sufficient for purposes of illustration.

If, then, any one of the ocular motor functions may be involved independently of the rest, we must next inquire what may be **the nature of the lesion** affecting it. In answer to this it may be said that any motor function may be deranged in either one of three ways.

(1) It may be performed inadequately or not at all—deficiency of movement *(Hypokinesis).*

(2) It may be performed excessively—excessive movement *(Hyperkinesis)*.

(3) It may be performed irregularly or in successive phases of excess and inadequacy—irregular movement *(Parakinesis)*.

To the question whether disorders having these various characters are actually encountered in connection with the ocular movements, clinical facts once more enable us to give an affirmative answer. It must, however, be understood that these conditions, namely hypokinesis, hyperkinesis and parakinesis, may be present in very **varying degrees.** Thus weakness (hypokinesis) may vary all the way from a slight and transient enfeeblement to a complete paralysis; and, similarly, excessive action (hyperkinesis) may range from a moderate degree of over-action to an intense and permanent spasm. It thus happens that there are some deviations so slight as to be habitually corrected by the supplementary effort that the patient is able to exert *(Superable deviation, Latent strabismus, Heterophoria)*; while there are other deviations so great that the patient can overcome them with difficulty if at all, and which are hence more or less constantly present *(Insuperable deviation, Manifest squint, Heterotropia)*. These latter again are divided according to the constancy of their occurrence into *Intermittent*, when present at intervals, *Periodic*, when recurring regularly under certain conditions (*e. g.* in convergence), and *Constant*. It should be borne in mind, however, that these distinctions all represent differences simply of degree and not of kind.

The foregoing considerations, confirmed and modified by the results of the examination of quite a large amount of clinical material, have led me to propound the following **classification of the motor anomalies of the eye.**

CLASSIFICATION OF THE MOTOR ANOMALIES OF THE EYE. OCULAR DEVIATIONS IN GENERAL DUE TO

I. Anomalies of Individual Muscles or of their Nerve-nuclei.
   (*a*) *Under-action.* The muscle works inefficiently.
      1. Because the muscle itself is ill-developed or atrophied (Structural Squint), or because its attachments are unfavorable for effective action (Insertional Squint).

2. Because of impairment of the nerve or nerve-nucleus supplying the muscle (Paretic Squint).

(*b.*) *Over-action.* The muscle works excessively.

1. Because the muscle itself is over-developed (Structural Squint), or because its attachments are favorable for effective action (Insertional Squint).

2. Because of over-excitation of the nerve or nerve-nucleus supplying the muscle (Spastic Squint).

(*c.*) *Perverted action.* Clonic spasm of individual muscles (some rare forms of Nystagmus).

II. Anomalies of the Association Centres for Parallel Movements.

(*a.*) *Under-action.* Producing an equal impairment in the movement of both eyes either (1) up, (2) down, (3) to the right, (4) to the left, or (5) obliquely, or (6) an equal impairment of the rotary (swivel) movements of the two eyes. (Associated Paralysis, Conjugate Paralytic Deviation.)

(*b.*) *Over-action.* Producing an equal excessive movement of both eyes in the same direction (Associated Spasm, Conjugate Spastic Deviation).

(*c.*) *Perverted action.* Clonic Spasm (Ordinary Nystagmus; including Lateral, Vertical, Rotary and Mixed Nystagmus).

III. Anomalies of the Centre for Convergence-Movements.

(*a.*) *Under-action.* Convergence-Insufficiency (producing one variety of Comitant Divergent Squint or Exophoria).

1. Accommodative (due to relaxed accommodation in myopes).

2. Non-accommodative.

(*b.*) *Over-action.* Convergence-Excess (producing one form of Comitant Divergent Strabismus or Esophoria).

1. Accommodative (due to excess of accommodation in hypermetropes.)

2. Non-accommodative.

IV. Anomalies of the Centre for Divergence Movements.

(*a.*) *Under-action.* Divergence-Insufficiency (producing one form of Comitant Convergent Squint or Esophoria).

(*b.*) *Over-action.* Divergence excess (producing one form of Comitant Divergent Squint or Exophoria).

V. Anomalies of Sursumvergence.

(*a.*) *Under-action.* Sursumvergence-Insufficiency.

(*b.*) *Over-action.* Sursumvergence-Excess (producing constant or intermittent vertical divergence of the visual lines; including cases in which one visual line is habitually above (Spastic Right or Left Hyperphoria), and cases in which sometimes one, sometimes the other, rises higher (Alternating Hyperphoria).

VI. Anomalies of Rotation (Swivel or Torsion) Movements.

(*a.*) Habitual or intermittent divergence of the Vertical meridians (Cyclophoria Divergens).

(*b.*) Convergence of the vertical meridians (Cyclophoria Convergens).

VII. Mixed Forms. Many varieties including particularly—

(*a.*) Convergence-Insufficiency combined with Divergence-Excess.

(*b.*) Convergence-Insufficiency combined with Divergence-Insufficiency.

(*c.*) Convergence-Insufficiency, simple or complicated, combined with under-action or over-action of one or more of the lateral or vertical muscles.

(*d.*) Convergence-Excess combined with Divergence-Insufficiency.

(*e.*) Convergence-Excess combined with Divergence-Excess.

(*f.*) Convergence-Excess, simple or complicated, combined with under-action or over-action of one or more of the lateral or vertical muscles.

(*g.*) Under-action of one muscle combined with over-action of another.

Before taking up the study of the individual anomalies, it is well to consider one or two features common to all of them.

Comitant* and Non-comitant Deviations. A wide difference exists both as regards symptoms and physical signs between motor disorders in which the amount of deviation constantly changes according to the direction of the gaze *(Non-comitant deviations)* and those in which it remains the same *(Comitant deviations)*. The former is apt to be much the more troublesome of the two to the patient, particularly when the deviation keeps changing while the eyes are executing associated parallel movements. This is apparently due to the fact that the symptoms occasioned by the deviation, changing as they do with every alteration in the position of the eyes, cannot be allowed for nor be as readily ignored as when they are constant.

That is, since the patient sometimes sees single, sometimes double, *i. e.* is affected with an error of varying amount, he cannot as readily make allowances for his peculiar state of vision as if he saw double all the time and had a constant error to contend with. Moreover, it is less easy for the eye to suppress a false image when the latter, instead of occupying always the same spot upon the retina, as it does in comitant deviations, occupies a number of different spots in succession, as it does in the case of a non-comitant deviation. Hence the diplopia tends to persist much longer in non-comitant disorders than in those that are comitant. Besides, the very fact that the deflection is changeable in amount is the cause of the most trying symptoms (false projection and vertigo) of a non-comitant deviation; and

*The word "comitant" is here employed in preference to the more usual "concomitant" as being both briefer and etymologically better.

when the deflection becomes constant in quantity for all directions of the gaze, these symptoms are apt to disappear.

It is obvious that *an affection of the individual ocular muscles*, whether in the direction of excess or of deficiency, must cause a non-comitant deviation, the angle between the two visual lines becoming greater and greater the more the eyes are carried in the direction in which the muscles in question normally exert their greatest influence. Thus in a paralysis of the right externus the right eye will lag more and more behind its fellow in proportion as the eyes are carried to the right; and in paralysis of the right superior rectus the right eye will fall more and more below the level of the other, the more the attempt is made to direct the gaze up and to the right. In fact, it is by this very changeableness or non-comitancy of the deviation that we make our diagnosis of the existence of muscular paralysis or spasm.

On the other hand, a deviation due simply to an over-action or under-action of either *divergence* or *convergence* will not change in the performance of associated lateral or vertical* movements, as long as the object of fixation does not appreciably approach or recede from the eyes. For, for any given distance, the amount of convergent or of divergent action will be constant and the excess or deficiency of this action will also be constant, no matter whether the eyes are looking straight ahead, or laterally, or up or down. If, however, the distance of the object of fixation from the eye is altered, the amount of convergent or divergent action changes also, and in general the amount of deflection will change as well. Thus in a pure convergence-insufficiency the deviation will become more and more apparent as the limit of convergence is approached and will disappear altogether when the convergence is relaxed, *i. e.* when the patient is looking at a distance. In the case of a divergence-insufficiency the reverse will hold good.

In *anomalies of associated parallel movements* the motions of the eyes are strictly comitant, and in fact the visual lines remain everywhere parallel, since the ocular movements, while either restricted or in excess in some given direction, are restricted or in excess to the same degree in both eyes. For example, in a case of restricted sursumversion both eyes will fail to move up, but as the failure affects both to an equal degree, the visual lines will in the performance of this movement remain parallel up to the point where they cease to move at all.

The foregoing facts may be recapitulated as follows:

(1) *A deviation which increases or decreases in the performance of associated parallel movements by the eyes signifies an anomaly of one or more of the ocular muscles* — the

*This statement is not absolutely accurate, for, owing to the fact that the visual lines tend to diverge when the gaze is directed upward and to converge when they are directed downward, an exophoria otherwise comitant will show an increase in the upper and a decrease in the lower portions of the field of fixation.

direction in which it increases corresponding to that in which the action of the muscles affected is normally most pronounced.

(2) *A deviation which remains constant or nearly so while the eyes are performing parallel movements is due, not to an anomaly of individual muscles, but to an anomaly of some one of the associated movements of the eyes.*

(3) *A deviation which increases as the eyes are converged denotes a convergence-anomaly, and one which increases as the eyes are passing from convergence to parallelism a divergence-anomaly.*

Transformation of Non-comitant into Comitant Deviations. Non-comitancy in parallel movements might be remedied by restricting or increasing the action of the unaffected eye in the same sense as that in which the action of the affected eye is restricted or increased; *i. e.* by converting the anomaly from one of Class I. into one of Class II.

This is done in actual practice when, *e. g.* in a paralysis of the superior oblique of the right eye we tenotomize its associated antagonist, the inferior rectus of the other eye, thereby weakening the latter artificially in precisely the same sense and to the same extent that the right eye is weakened naturally. In nature a similar, though less perfect, result is commonly attained by the development of a spasm of the direct antagonist or of a pair of antagonistic muscles in the eye affected. Thus in a paralysis of the right externus, which produces a deviation confined to the right half of the field of fixation, is usually followed after a time by a spastic contraction of the right internus, which produces a deviation of the same character in the left half of the field of fixation. Thus the affected eye gets to squint inward, not only when looking to the right, but also when looking to the left, and the deviation, from being markedly non-comitant, becomes comitant or nearly so. A similar occurrence is regularly observed in paralysis of the other muscles.

A similar tendency to replace non-comitant by comitant deviations appears to prevail, although possibly to a less extent, in divergence and convergence anomalies. Thus a convergence-insufficiency, existing at first without any complication, is very apt later on to become associated with a divergence-excess, so that a sensibly constant deviation outward (exophoria) is present both for far and near, where originally it was present for near only. So also a convergence-excess may lead to a divergence-insufficiency, so that the esophoria which at first was marked only for near now becomes equally pronounced for distance. This compensatory process, by which comitancy is evolved

out of a non-comitant state, is doubtless the reason for the many mixed forms of divergence and convergence anomalies that we meet with; and the process itself may actually be watched in following the development of many cases of squint.

The process in fact seems to take place so generally that it seems safe to enunciate the following law:

A non-comitant deviation usually tends to become comitant, there being superadded to the morbid condition already existing another by means of which the former is generalized and rendered sensibly equal throughout the whole field of fixation.

V.

ANOMALIES OF THE INDIVIDUAL MUSCLES.*

Hypokineses and Hyperkineses (Paretic and Spastic Squint). Over-action or under-action of the individual ocular muscles, giving rise to a disturbance of the normal balance of the eyes, may be due to

(a) Over or under-development of the muscle itself (structural squint, structural heterophoria).

(b) Variations in the origin, insertion and direction of the muscles and in the length of their tendons (insertional squint or heterophoria).

(c) Over or under-excitation of the muscle due to some affection of its nerve or nerve-nucleus (paretic and spastic squint and heterophoria).

The hypertrophy or the non-development of the muscles producing what I have called **structural deviation** is probably in many cases *congenital*. This is certainly so in those cases of defective elevation of the eye (frequently associated with ptosis) which have been proved by dissection to be due to absence of the superior rectus (Fuchs). I have seen one marked instance of this congenital anomaly in which ptosis co-existed with almost entire absence of elevation, and I am inclined to think that several other cases of less complete paralysis of elevation, which I have

*This section, since its first presentation to the Prize Committee, has been largely added to and to a certain extent re-modeled.

observed, were likewise congenital and structural in origin.*

Another class of cases of structural deviation, of quite frequent occurrence, are those which Schneller (Arch. für Ophth. xxiii. No. 3) describes under the name of *muscular strabismus*. Such, for example, are those cases of divergent strabismus in which, at the time of operation, we find the external rectus thick, broad and fleshy, and provided with a dense, broad tendon, while the internal rectus, on the contrary, is thin and has a narrow tendon, often split up into separate fibrils. Schneller in these cases found that the tendon of the externus, instead of being about equal to that of the internus†, was from one-fourth to one-third greater. In convergent strabismus, on the other hand, the ratio was reversed, the tendon of the externus being much thinner than that of the internus and the externus itself being comparatively meager and undeveloped.

Schneller reports thirty-four cases of such muscular (structural) strabismus. He regards them (probably with justice) as congenital in origin.

Others have supposed that these were cases of acquired atrophy (atrophy of disuse). But the pathological changes are those of simple non-development rather than atrophy, the muscular fibres being merely insufficient in number or size and showing no evidences of fatty or fibrous transformation nor of myositis.

The diagnostic features of a muscular squint, according to Schneller, are

(1) The deviation is not relieved by atropinization and correction of the refraction (distinction from accommodative convergence-excess and convergence-insufficiency).

(2) The field of fixation (*i. e.* the range of excursion of the eyes) is abnormally large in one direction and abnormally limited in the opposite. If the sum of the inward excursions of the two eyes exceeds the sum of the outward excursions by 30°, a convergent strabismus is produced; while a preponderance of outward excursions over inward excursions of 12° is sufficient to cause a divergent squint (Schneller).

*See article by the author on "Paralysis of the Superior Rectus and Its Bearing upon the Theory of Muscular Insufficiency" (Archives of Ophthalmology, vol. xxiii., No. 1, 1884.)

†Schneller found the normal ratio between the breadth of the externus-tendon and the internus-tendon to be 102:100. His measurements were made so as to include more of the scleral attachments than in those made by other observers who have found the ratio to be 88 (Volkmann) and 89 (Fuchs).

This latter statement involves a fallacy, strictly speaking, if the range of excursion is, as is usually the case, determined by making the eyes follow a test-object situated within a foot or so of the eye. For in this case when the eyes are directed to the right, the amount of excursion outward of the right eye = the absolute amount by which the externus can turn it outward (abduction) less the amount by which it is turned inward in order to converge upon the object (convergence). The latter (convergence) movement will be excessive either if the right internus acts too forcibly in response to an ordinary impulse of convergence (muscular excess) or if, the muscle being normal, the impulse itself is excessive (convergence-excess). In either case the net movement outward will be limited, even when the actual amount of abduction produced by the externus is normal. If, then, the case were one of simple convergence-excess the limitation of outward movement would be the same for both eyes, *i. e.*, the same whether the gaze is directed to the right or to the left: but if it were one of muscular squint, *i. e.* of actual insufficiency of the externi (or preponderance of the interni) the limitation might be either unilateral or bilateral. In the latter event the diagnosis of a muscular anomaly, as distinguished from a pure anomaly of convergence, would be substantiated if the limitation of abduction were found to be the same for all distances, far as well as near.

Degrees of weakness or of over-development, too slight to produce actual squint, probably form an important element in the causation of many cases of heterophoria *(structural heterophoria.)*

Insertional squint or heterophoria, *i. e.* that sort of muscular under-action or over-action due to variations in the origin and insertion of the tendons, is difficult to distinguish from the structural deviations due to under or over-development of the muscular fibre itself. The structural deviations, however, are frequently congenital, while insertional squint is generally acquired. For example, a very important class of cases coming under the latter category are those in which the insertion of a tendon has been displaced by a *tenotomy* or an *advancement*. Here by the operation an insertional anomaly is produced: and the weakness or over-action resulting from this artificial anomaly is precisely similar in symptoms and objective signs to weakness or over-action due to natural causes (paresis or spasm, for example).*

*Of the exactness of this similarity I have been able to convince myself by repeated examinations upon cases in which extensive operations had been made either upon the lateral or vertical muscles.

Another class of cases in which there is an acquired insertional anomaly are those in which a *divergent strabismus has developed as a result of the gradual divergence of the orbits* taking place during the period of growth in childhood and youth. This divergence, by altering the angle of insertion of the tendons and the amount of tendon in contact with the eye-ball, increases very greatly the power of the externus and diminishes that of the internus, and in itself is quite sufficient to account for the development of the strabismus (see L. Weiss, Arch. f. Augenh. xxix. and Arch. of Oph. xxv. No. 3, 1896). The anomaly in this case is of great importance in that it exaggerates and renders uncertain the effect of a tenotomy of the interni when made upon children. Admitting Weiss's explanation of it, the obvious deduction would be that tenotomy of the interni should be avoided in children with convergent squint who have the orbits and eyes set very close together, since in these divergence of the orbits is likely to take place subsequently resulting in a preponderance of the externi, which will by itself suffice to correct the convergence.

Other kinds of insertional anomalies exist, some so slight as to produce only a moderate degree of *heterophoria*, instead of a squint. They often develop when a non-comitant deviation has lasted for some time, and help to transform the latter into a deviation of the comitant variety.

Muscular paresis and spasms (Innervational Deviations). The subject of the **paralyses** of the eye muscles has been so thoroughly worked out and so ably presented by the two Graefes and Mauthner, that there is but little to add to their statement. The few points to which it seems necessary to call attention will be touched upon in the remarks on diagnosis.

Spasm of the individual ocular muscles may occur

(1) As an *evidence of a secondary deviation in a sound eye* when the other eye, being paretic, is yet used for fixation.

Thus suppose that a patient with a paresis of the right externus nevertheless fixes with the right eye because he sees better with it.

Then when he looks at some object situated on his right he has to exert an excessive amount of force to turn his fixing eye out sufficiently. The same amount of force is, by the law of association, also transmitted to the left internus, and the latter, not being paretic, will respond with normal vigor to the excessive stimulus, so as to carry the left eye much further to the right than its fellow, and hence quite to the right of the object of fixation. In this case, as the right eye follows the object, and the left eye by an apparently excessive action shoots past it, we might regard the condition as one of veritable spasm of the left internus; whereas the real state of the case is that the latter muscle is normal and contracts excessively only because the stimulation that it receives is excessive. These cases are therefore correctly denominated as cases of *false* or *apparent spasm*.

(2) In the case of a paralysis of an ocular musle, *a spastic contraction of one or more of the other muscles in the same eye* takes place, by virtue of which, as has been already stated, the deviation becomes more or less concomitant and generalized throughout the whole field of fixation. This contraction finally results in a permanent shortening (contracture) of the muscles affected, but before this stage has been reached, a temporary and varying spasm may be set up in the antagonists of the paralyzed muscle, so as to carry the deviation and the diplopia far outside of the field of action of the latter. This was clearly shown in the following case which came under my care:

Paralysis of Inferior Rectus. Varying Spasm of Superior Rectus of same eye. H., male, aged 29. Vertigo, blurring of sight, and diplopia for past two years. No history of syphilis. Presents all the evidences (by inspection, screen, and diplopia tests) of a well-marked paresis of the R. inferior rectus. Vertical diplopia (with image of right eye below = DR) increasing to 6° to 7° (= 14° prism) as the eyes are carried down and to the right; diminishing to zero when the gaze was directed down and to the left. Moderate crossed diplopia everywhere; increasing when eyes are directed down and to left. Vertical diplopia of the same character as that found in the lower field prevailed, although to a less extent in the upper field; most marked in looking up and to the right (Eu & r, DR increasing). This diplopia in the upper field varying much, and partially superable by voluntary effort.

Here there was evidently a spasm (variable in amount) of the right elevators, and particularly of the right superior rectus (shown by the fact of the vertical diplopia increasing upward and to the *right*). The vertical diplopia in the upper part of the field might, it is true, have been due to a paresis of the elevators of the left eye; but the

variable character of the diplopia and the fact that it was superable by voluntary effort negative this supposition.

(3) Spasm (usually slight) of one or more of the ocular muscles may occur as the result of *irritative lesions at the base of the brain*, particularly meningitis.

(4) Slight transient spasm may occur in *chorea*.

(5) Convulsive tonic spasm may occur in *epilepsy* and *hysteria*, although in these diseases it is not generally the individual muscles that are affected, but the associated movements of the eyes (particularly the parallel movements and movements of convergence). Gowers, however (Diseases of the Nervous System), calls attention to a sort of convulsive seizure that may be styled epileptoid, in which there are suddenly developing tonic and clonic spasms of one or more ocular muscles, associated with more or less obtunding of the consciousness.

(6) Finally we may have cases of *non-paroxysmal and chronic spasm of some one ocular muscle*, which causes the eye to make an excessive movement as soon as it is turned so that the affected muscle can act upon it. Thus cases have been described in which a spasm of the external rectus caused the eye to shoot far outward as soon as the eye was turned past the middle line. Mauthner (Augenmuskellähmungen) casts doubt on all such cases, regarding them as probably instances of secondary deviation of a sound eye due to the fact that the other eye is paretic but still performs fixation, so that an excessive impulse is transmitted to both eyes. That is, he would class all such cases under the category of false or apparent spasm (Group 1 mentioned above). But this statement is certainly too sweeping, since cases of chronic spasm, although doubtless rare, do occur, as witness the following that I observed:

Paralysis of L. Externus; Spasm of L. Inferior Oblique. Bertha S., aged 8. Deviation of eyes noticed since birth. Used to hold head to right and does so still when looking intently at anything. L. eye cannot move out beyond median line. When eyes are carried horizontally to the right, *L. eye suddenly flies upward and becomes buried beneath the upper lid.* In associated movements in lower field, L. eye moves normally with the R. Behind screen L. eye deviates high up and somewhat in. Deviation of either eye behind screen about equal. No double images attainable. V. R. $^{20}/_{40}$: with +1.00 D. $^{20}/_{30}$. L. $^{20}/_{70}$.

Here the sudden and excessive upward movement of the left eye when directed inward could only have been effected by a spasmodic contraction of the inferior oblique; the superior rectus acting but slightly as an elevator when the eye is adducted.

A similar case in which with **paralysis of the R. externus and R. superior rectus** there was **spasm of the R. superior oblique** is the following:

Emma D., aged 15. Strabismus since scarlatina eleven years before. Occasional vertigo; no diplopia. Movements of L. eye normal. R. eye cannot move at all to right, nor upward and to right: movement upward almost normal when eyes are carried to left (retention of power of inferior oblique); movement downward excessive, especially when eyes are directed to left (excessive action of superior oblique); when attempt is made to move eyes straight to left, R. eye shoots obliquely down and to the left (spasmodic action of superior oblique).

In addition to these cases in which spasm was associated with paralysis, I have seen others in which a deviation limited to one portion of the field of fixation was present, which deviation was always marked but yet *varied so much in amount* from one time of observation to another as to necessitate the assumption of its being spasmodic rather than paralytic in origin.

Symptoms and Differential Diagnosis of the Different Varieties of Muscular Over-action and Under-action. No attempt will be made here to go at all fully into the symptoms produced by insufficiency or over-action of the individual muscles. This has already been done in various excellent treatises. I will simply give the following facts to which, as I conceive, too little attention has been paid.

1. In the deviations considered in the foregoing paragraphs the abnormality, as has been pointed out, may affect either the muscle itself, its tendon, or the nerve and nerve-nucleus supplying it; *i. e.* the muscle may act above or under the normal either because it is itself too much or too little developed (Structural Deviation); or because the direction and insertion of its tendons are not what they should be (Insertional Deviation); or because the muscle is in a state of paralysis or spasm (Innervational—Paretic or Spastic—Deviation). Now it cannot be too strongly insisted upon that *these three varieties, however dissimilar*

in origin, show in their symptoms and objective signs no real point of difference. Thus weakness of the external rectus, whether arising from feeble and arrested development of the muscular fibre, or from mal-position of the tendon (natural or produced by a tenotomy), or from lesion of the sixth nerve, gives rise to the same symptoms and offers the same appearances to objective examination. The characters of the paretic and spastic deviations, to be sure, are generally more definite and more striking than are those of the other anomalies mentioned, but this is simply owing to the fact that such cases are usually seen early, before any compensatory changes have taken place and while the diplopia, false projection, vertigo, and other symptoms are therefore still marked. Precisely the same effect will be produced in a patient with binocular single vision if one of the tendons is completely divided. And in cases of long standing—particularly in congenital cases—it is impossible to say with certainty from the symptoms alone without reference to the history whether faulty innervation, faulty insertion, or faulty structure lies at the basis of the anomaly.

2. *Congenital deviations* present several peculiarities. In the first place they cause little or no trouble for the obvious reason that the patient from his infancy has learned to adjust himself to the anomalous condition present. Again, it is remarkable that, even after the condition has existed for years, there is no constant suppression of the diplopia such as occurs in comitant squint, and furthermore, there is no tendency, as in the case of other non-comitant deviations, to a transformation into a comitant deviation by the development of a contracture of the antagonists. The reason probably is that as the patient has never known any different condition from that which he was born with, and as this condition consequently is the natural one for him, he experiences no inconvenience from its continuance, so that no involuntary tendency is set up toward its rectification. The result is that these congenital deviations, which may be due either to paresis or to a structural defect, afford, as far as objective signs go, the most typical picture of uncomplicated muscular paralysis that we can have.

3. *Acquired muscular deviations*, on the other hand, whether due to anomalies of innervation or insertion, always tend to become *comitant;* this being affected by a gradually developing contracture of the antagonists when the affected muscle is too weak, and by a weakening of the antagonists when the affected muscle is too strong. It is thus, probably, that many cases of comitant strabismus convergens and strabismus divergens are produced; and the few cases of strabismus sursumvergens that I have met with were apparently all developed in this way.

Thus paralysis of the R. elevators, when not of congenital origin, is regularly followed by spasm of the R. depressors, causing the right eye to stand lower than the other in all portions of the field of fixation. This process continuing and the spasm developing into a confirmed contracture, the difference in height between the two eyes (L. hypertropia) becomes equal for all directions of the gaze; the limitation of movement of the right eye upward is compensated for by a corresponding increase in its movements downward; and the picture of a typical comitant *strabismus deorsumvergens* is produced.

If but one of the elevators, *e. g.* the superior rectus, is paretic the spasm is apt to be confined, in the first instance at least, to the corresponding depressor, *i. e.* the inferior rectus. In this case the deviation will become half-comitant, that is, comitant for movements up and down but not for lateral movements. Thus, if the right superior rectus were the paretic muscle, we should have first a L. hyperphoria, very marked in the upper right-hand quadrant of the field of fixation and diminishing rapidly, both when the eyes were carried down and when they were carried to the left. Later, when spasm of the right inferior rectus had developed, the L hyperphoria would become pronounced in the lower right-hand quadrant also, and ultimately so much so that whether the eyes were carried up or down, the right eye would always stand about the same distance below the left. But in both the upper and lower portions of the field the L. hyperphoria) would still increase as the eyes were carried to the right and diminish to zero as they were carried to the left: so that even under these circumstances the deviation would not be comitant for lateral movements.

A case of this sort, in which the inferior rectus was the muscle primarily affected and in which semi-comitancy was being developed by spasmodic action of the superior rectus has already been mentioned (under the head of Spasm of the Ocular Muscles).

Cases in which a comitant (and even an alternating) *lateral squint* has developed out of a paresis of the externus, followed by contracture of the internus and later by partial restoration of the power of

the externus, have been reported by Spicer ("Royal London Ophth. Hosp. Reports," xiv. 1, 1895). I have observed analogous cases, particularly one in which a partial oculomotor paralysis with secondary contracture of the externus had resulted in a nearly comitant strabismus divergens.

In proportion as the deviation becomes transformed from a non-comitant to a comitant one, the symptoms grow less disturbing; for, as already stated, it is always the case that, other things being equal, a comitant deviation will give less trouble than will one that is non-comitant.

4. The *diagnosis between over-action and under-action* is often difficult. Theoretically, over-action (spasm) is associated with excessive movement, and under-action (paresis) with very deficient movement in some one direction. But, unless the paralysis is nearly total or the spasm is great, it is not always easy to say whether it is the muscle of one eye that is under-acting or whether it is the associated antagonist of the other eye that is over-acting; and when secondary changes have taken place, transforming the deviation into a comitant one, the diagnosis is often impossible.

Before this has occurred the differentiation may be made from the following considerations.

OVER-ACTION.	UNDER-ACTION.
Points in Common.	
One eye moves faster and further than the other when both are carried in some one particular direction; and this discrepancy between the position of the eyes and also the diplopia, false projection, and vertigo become more and more pronounced, the further the eyes are carried in that direction. The primary position for both eyes is nearly the same.	
The absolute excursion of the faster moving eye in the given direction is greater than normal; that of the other eye is normal.	The absolute excursion of the faster moving eye is normal in all directions; that of the other eye is subnormal.
The total excursion of the faster moving eye in the given direction and in its opposite is greater than normal; *i. e.*, the field of fixation is excessively large in one of its diameters.	The total excursion of the faster moving eye is normal, that of the other eye is subnormal; *i. e.*, the field of fixation of the latter is contracted in one of its diameters.
Fixation is usually performed by the slower moving eye.	Fixation usually performed by the faster moving eye.
The amount of deviation may show great and sudden changes from time to time.	The amount of deviation remains constant or changes slowly and progressively.

5. While the diagnosis of a marked underaction of one of the ocular muscles usually presents no difficulty, there is quite a large number of cases in which the condition is not so obvious, the *weakness of the muscles being comparatively slight.** In these cases diplopia does not occur until the eyes are removed some distance (10° to 20°) from the primary position. As soon as it does appear, however, it develops in a perfectly typical fashion, showing marked increase in some one special direction and diminishing elsewhere to zero. Most cases of this sort of anomaly, that I have observed, have been those in which there was a pretty pronounced weakness (perhaps of congenital origin or, rather, due to non-development) of the superior rectus; less frequently the inferior rectus seemed involved. The nature and course of these cases have not been thoroughly investigated, but it is noticeable that they are frequently associated with a convergence-insufficiency which shows a tendency to go over into a divergence-excess, resulting, in sone instances at least, in a regular divergent squint. The chief symptoms (which, however, are, very likely, attributable to the convergence-insufficiency) are conjunctival irritation, asthenopia (often very marked), headache, and diplopia. In many cases the symptoms are insignificant and this fact coupled with the absence of diplopia in the primary position is probably the reason why these cases have been left uninvestigated. Quite a number of cases of hyperphoria doubtless belong to this category.

Another variety of muscular weakness, transitory in its character, is that already referred to as associated with *neurasthenia*. Here we may find diplopia all round the periphery of the field of fixation, *i. e.* within 35° of the primary position (Concentric contraction of the field of binocular single vision). This condition would appear to have little significance except as a diagnostic sign of the condition with which it is associated.

It is not unlikely that *slight degrees of muscular spasm* are at the bottom of some of the cases of heterophoria that we meet with; *e. g.* spasm of either one of the elevators

*It is to these cases that the old term muscular insufficiency should be limited.

or of either one of the depressors may occasion a hyperphoria of more or less varying character *(Spastic heterophoria)*. I have notes of five or six cases in which the presence of a varying and even alternating hyperphoria seemed to be due to more or less spasmodic contraction of the vertical muscles.

Finally it is quite likely that a large number of cases of slight deviation are due to *moderate under-action of one muscle combined with over-action* (secondary spasm) of another, so as to produce a more or less comitant deflection. Probably most of the cases of hyperphoria, in which the deviation, although moderate in amount, is present through the whole field of fixation, are attributable to combined anomalies of this sort *(comitant hyperphoria)*. Cases of this kind often give rise to asthenopia, headache, neuralgia, and sometimes to gastric disturbance and impairment of nutrition. Diplopia is infrequent and when present can usually be readily overcome.

6. The diagnosis of the muscles affected in a case of paralysis or spasm is readily *accomplished by means of the double images* if the following principles are kept in mind.

(a) The diplopia increases progressively as the eyes are carried in that direction in which the action of the muscle affected is normally most pronounced.

(b) Paralysis of any given muscle of one eye produces diplopia of the same character and increasing in the same way as does a spasm of the associated antagonist in the other eye.

(c) While paresis or spasm of one of the vertical muscles does produce lateral diplopia, this diplopia may be and often is neutralized by the existence of other factors, so that it is of no practical significance in the diagnosis. The latter must rest solely upon the difference in height of the double images and the portion of the field where this difference is most pronounced.

This fact, enunciated and explained very clearly by Mauthner, is frequently overlooked, many insisting that paresis of the superior and inferior recti must produce crossed, and paresis of the obliques homonymous diplopia. But, apart from the fact that these pareses are often associated with exophoria or esophoria of sufficient

amount to neutralize their lateral diplopia, the natural tendency which the visual axes display to diverge when the eyes are directed up and to converge when they are directed down will work to abrogate the homonymous diplopia due to a paresis of the inferior oblique and the crossed diplopia due to a paresis of the inferior rectus. As a matter of fact I have found paresis of the superior and inferior recti often associated with homonymous diplopia.

The following table, constructed in accordance with the three principles just enunciated, shows succinctly the way in which the diagnosis can be made from the double images.

DIAGNOSTIC TABLE

of Paralysis and Spasm of the Ocular Muscles.

NOTE. DII = homonymous diplopia; DX = crossed diplopia; DR = vertical diplopia with the image formed by the right eye below; DL = vertical diplopia with the image formed by the left eye below. Eu, Ed, Er, El, Eu & r, etc., = associated parallel movements in which the eyes are directed respectively up (Eyes up) down, right, left, up and right, etc. ≻≻ = increasing; ≺≺ = decreasing.

DIPLOPIA		= PARESIS OF	OR = SPASM OF
Er. DII	≻≻ greatly.	R. External Rectus	L. Internal Rectus
Er. DX	" "	L. Internal Rectus	R. External Rectus
El. DII	" "	L. External Rectus	R. Internal Rectus
El. DX	" "	R. Internal Rectus	L. External Rectus
Eu. & r. DL	" "	R. Superior Rectus	L. Inferior Oblique
Eu. & r. DR	" "	L. Inferior Oblique	R. Superior Rectus
Eu. & l. DR	" "	L. Superior Rectus	R. Inferior Oblique
Eu. & l. DL	" "	R. Inferior Oblique	L. Superior Rectus
Ed. & r. DR	" "	R. Inferior Rectus	L. Superior Oblique
Ed. & r. DL	" "	L. Superior Oblique	R. Inferior Rectus
Ed. & l DR	" "	R. Superior Oblique	L. Inferior Rectus
Ed. & l. DL	" "	L. Inferior Rectus	R. Superior Oblique

The above table is sufficient for all ordinary working purposes, but for the sake of completeness I give also the more extensive one subjoined, which I have devised to show how the character of the diplopia may be modified by circumstances.

TABLE OF DIPLOPIA.

A. HOMONYMOUS DIPLOPIA VARYING WITH DIFFERENT DIRECTIONS OF THE GAZE.

I. Variation great = Paresis of Externus (Spasm of Internus.)

CHARACTER OF DIPLOPIA	INDICATES PARESIS OF	OR SPASM OF
DII ⋗⋗ in Er, ⋖⋖ in El.	R. External Rectus	L. Internal Rectus
DII ⋗⋗ in El, ⋖⋖ in Er.	L. External Rectus	R. Internal Rectus

II. Variation slight = Paresis of Oblique (Spasm of Superior or Inferior Rectus) or a comitant esophoria associated with a condition causing varying DX.

DII ⋗⋗ in Er.	Particularly marked in Eu. [Eu. & r.]	R. Inferior Oblique	L. Superior Rectus
		R. Superior Rectus	L. Inferior Oblique
	Particularly marked in Ed. [Ed. & r.]	R. Superior Oblique	L. Inferior Rectus
		R. Inferior Rectus	L. Superior Oblique
DII ⋗⋗ in El.	Particularly marked in Eu. [Eu. & l.]	L. Inferior Oblique	R. Superior Rectus
		L. Superior Rectus	R. Inferior Oblique
	Particularly marked in Ed. [Ed. & l.]	L. Superior Oblique	R. Inferior Rectus
		L. Inferior Rectus	R. Superior Oblique

B. CROSSED DIPLOPIA VARYING IN DIFFERENT DIRECTIONS OF THE GAZE.

I. Variation great = Paresis of Internus (Spasm of Externus.)

CHARACTER OF DIPLOPIA	= PARESIS OF	OR SPASM OF
DX ⋗⋗ in Er., ⋖⋖ in El.	L. Internal Rectus	R. External Rectus
DX ⋗⋗ in El., ⋖⋖ in Er.	R. Internal Rectus	L. External Rectus

II. Variation slight = Paresis of a Superior or Inferior Rectus (Spasm of an Oblique) or a comitant exophoria associated with a condition causing varying DII.

DX ⋗⋗ in Er. ⋖⋖ in El.	Particularly marked in Eu. [Eu. & r.]	L. Superior Rectus	R. Inferior Oblique
		L. Superior Oblique	R. Superior Rectus
	Particularly marked in Ed. Ed. & r.]	L. Inferior Rectus	R. Superior Oblique
		L. Superior Oblique	R. Inferior Rectus
DX ⋗⋗ in El. ⋖⋖ in Er.	Particularly marked in Eu. [Eu. & l.]	R. Superior Rectus	L. Inferior Oblique
		R. Inferior Oblique	L. Superior Rectus
	Particularly marked in Ed. [Ed. & l.]	R. Inferior Rectus	L. Superior Oblique
		R. Superior Oblique	L. Inferior Rectus

C. VERTICAL DIPLOPIA VARYING IN DIFFERENT DIRECTIONS OF THE GAZE (= PARESIS OR SPASM OF ELEVATOR OR DEPRESSOR.)

I. Diplopia ⫘ in Eu. (Paresis or Spasm of Elevator.)

CHARACTER OF DIPLOPIA		= PARESIS OF	OR SPASM OF
DR ⫘ in Eu.	DR. most marked in Eu. & r. [regularly associated with DII ⫘ in Eu. & l.]	L. Inferior Oblique	R. Superior Rectus
	DR. most marked in Eu. & l. [regularly associated with DX ⫘ in Eu. & r.]	L. Superior Rectus	R. Iuferior Oblique
DL ⫘ in Eu.	DL most marked in Eu. & r. [regularly associated with DX ⫘ in Eu. & l.]	R. Superior Rectus	L. Inferior Oblique
	DL most marked in Eu. & l. [regularly associated with DII ⫘ in Eu. & r.]	R. Inferior Oblique	L. Superior Rectus

II. Diplopia ⫘ in Ed. (= Paresis or Spasm of Depressor).

DR ⫘ in Ed.	DR most marked in Ed. & r. [regularly associated with DX ⫘ in Ed. & l.]	R. Iuferior Rectus	L. Superior Oblique
	DR most marked in Ed. & l. regularly associated with DII ⫘ in Ed. & r.]	R. Superior Oblique	L. Inferior Rectus
DL ⫘ in Ed	DL most marked in Ed. & r. [regularly associated with DII ⫘ in Ed. & l.]	L. Superior Oblique	R. Inferior Rectus
	DL most marked in Ed. & l.regular associated with DX⫘ in Ed. & r.	L. Iuferior Rectus	R. Superior Oblique

From the foregoing table the following facts are apparent:

(1) *An homonymous diplopia, whether large or small, which increases as the eyes are carried to the right indicates under-action of* SOME *muscle of the right eye or over-action of* SOME *muscle of the left.*

(2) *A crossed diplopia which increases as the eyes are carried to the right indicates under-action of* SOME *muscle of the left eye or over-action of* SOME *muscle of the right.*

Treatment of Anomalies of the Individual Muscles. 1. In every case of over-action or under-action of the muscles our first attempts must be directed to removing the *cause* of

the trouble. This is effected with more or less success in syphilitic paralysis and in the paralysis caused by the pressure of meningitic effusions upon the nerves when we employ the iodides and mercury; in the so-called rheumatic paralysis, due apparently to exposure to cold, when we use sodium salicylate and the iodides; and when in hysterical disorders we employ the appropriate moral treatment.

2. When the cause is unknown or cannot be reached, an *expectant* treatment combined with *corroborant measures* (especially open-air exercise) must be employed. This is particularly the course to be pursued in spastic deviations, in hysterical and diphtheritic paralysis, and in the limitation of the movements of the eye due to neurasthenia.

The use of *strychnine*, advocated by some as being a direct tonic for insufficient muscles, seems to be efficient only so far as it acts to improve the patient's general condition. The same may be said of *electricity*.

3. When the deflection is slight, confirmed, and especially if it is more or less comitant, *prisms* often do good service, particularly in the vertical deviations.

In the lateral deviations they are of less service, because these are frequently complicated by the presence of divergence or convergence anomalies, in which the prolonged use of prisms is distinctly injurious.

4. *Operative measures* are to be adopted only when we are assured that the condition has reached a stage in which it is no longer likely to undergo change. Even in long-standing cases of paralysis it may be dangerous to operate, since the paralysis may disappear even after lasting for months, and then the operation which previously sufficed to correct the deviation will now over-correct or produce a deviation in the opposite sense which may be worse than the original one.

On the other hand, the judicious division of a contractured unparalyzed muscle may by giving the paralyzed muscle more play assist in restoring the power in the latter. See two cases by Chevallereau (Trans. of VIII. Internat. Ophth. Congress, Edinburgh, 1894) in which the movements of an eye the subject of a partial oculomotor paralysis were restored by tenotomy of the unparalyzed externus. These cases are analogous to those in which the power of the externi, weakened by persistent contracture of interni due to a convergence-

spasm, is gradually restored by the very prolonged use of the proper correcting glasses which relieve the spasm and allow the externi to act more freely. (See Long and Barret's cases, cited in section VII.)

In any case care must be taken that the *appropriate operation* is performed. The principles of operating, which were admirably enunciated by A. Graefe, may be thus stated:

Over-action of a muscle is to be relieved by tenotomy of the over-acting muscle. *Under-action* is to be relieved by tenotomy of the associated antagonist to the under-acting muscle, or, where this is impracticable, by advancement of the under-acting muscle itself. Sometimes, especially in paralysis of the externus and internus, these operations have to be further reinforced by tenotomy of the direct antagonist of the paralyzed muscle.

The reason for these rules is obvious. When a muscle is too strong we can reduce its action more or less effectively by a tenotomy. If the muscle is too weak, but yet retains some of its power, we can increase this power by performing an advancement. If, however, the muscle has lost all power, an advancement is obviously futile, and if the muscle acts but little an advancement alone is generally insufficient. Then we have only to remember that when a muscle is too weak, the effect upon the movements of the two eyes is the same as if the associated antagonist in the other eye was too strong; and if we tenotomize this antagonist, we shall limit the movement of the sound eye in the same sense and to the same extent as that in which the movement of the affected eye is already limited, *i. e.* both eyes will again move equally with each other, although the movement of neither will be normal. If no secondary changes have taken place, tenotomy of the associated antagonist alone may suffice, but usually the weakened muscle is opposed by a contractured and hence over-acting muscle in the same eye (direct antagonist), and therefore we may have also to divide the latter in order to secure the proper balance of action.

For the particular cases these rules may be expanded as follows:

Under-action of the R. externus indicates tenotomy of the L. internus, usually combined with advancement of the R. externus and often with tenotomy of the R. internus.

Under-action of the R. internus requires advancement of the R. internus combined with tenotomy of both externi.

The advancement in this case is imperatively demanded, because the internus has to be used in convergence as well as in associated

parallel movements. For the latter it might act sufficiently well, even if intrinsically weak, provided the externus of the other eye was also weakened and to the same extent: but for the proper performance of convergence movements the internus requires to be intrinsically strong, which can be effected only by an advancement.

Under-action of the R. superior oblique requires tenotomy of L. inferior rectus. This operation gives very satisfactory results.

Under-action of the R. inferior oblique requires tenotomy of the L. superior rectus.

Under-action of the R. superior rectus is in general best remedied by advancement of the muscle itself. If there is secondary contracture of the inferior rectus, the latter may be divided also, but this operation should be performed with considerable caution owing to the unpleasant effects produced by insufficiency of any one of the depressor muscles.

In very slight cases of insufficiency of the superior rectus in which the increase of the diplopia in lateral directions of the gaze would not be very great, and in which the aim is simply to remove the moderate hyperphoria in looking straight ahead, cautious tenotomy of the superior rectus of the other eye may replace the operation of advancement.

Under-action of the R. inferior rectus requires advancement of the muscle itself, which may need to be supplemented by tenotomy of the R. superior rectus.

Over-action of either one of the four recti is remedied by a tenotomy of the over-acting muscle.

Over-action of the R. superior oblique calls for advancement of the L. inferior rectus. In very slight cases this operation may be replaced by a cautious tenotomy of the R. inferior rectus.

Over-action of the R. inferior oblique is relieved by advancement of the L. superior rectus or, in very slight cases, tenotomy of the R. superior rectus.

It is probably from a failure to conform to these principles that a large part of the disappointment occasioned by the results of tenotomies done for heterophoria is due. For example, a L. hyperphoria due to insufficiency of a R. elevator muscle is treated by tenotomizing the R. inferior rectus. This operation does not essentially relieve the limitation of movement in the upper field, which really caused the hyperphoria and most likely the symptoms ascribed to the latter; on the contrary, it adds to the condition already existing a

new and very troublesome pathological condition, namely, a limitation of the movements of the right eye in the lower field also. In other words, to the paresis already present another paresis has been added by operation, and the patient suffers from the combined effects of both. That the effect of both pareses actually co-exist can readily be demonstrated by an examination of the double images. It will be found that the diplopia in the upper field has not been materially reduced by the operation, while a diplopia which did not exist before has been introduced into the lower field and gives rise to much disturbance. In this case advancement of the weak elevator in the affected eye or tenotomy of the elevator in the other eye would be the proper operation.

Parakineses. Tremor of Individual Ocular Muscles (Unilateral or Non-symmetrical Nystagmus). The tremulous movement (alternating spasm) that we denote by the name of nystagmus, although usually affecting equally the associated muscles in the two eyes, and therefore dependent in all probability upon a lesion of the association-centers, may be confined to the muscles of one eye or be of a different character in one eye from that in the other.

Hence, while ordinarily constituting a perversion of the associated parallel movements of the eyes, in these cases it must be regarded as a perversion of the movements of the individual muscles. These cases, are, to be sure, quite rare. Graefe (Graefe-Saemisch) reports four cases of unilateral vertical nystagmus (two observed by himself), and adds that, while he has seen cases of slight rotary nystagmus confined to one eye, he has never met with one of unilateral horizontal nystagmus. Frost, however (Trans. Ophth. Soc. of United Kingdom, xiv. 245), reports a case of acquired nystagmus in which the oscillations were horizontal in one eye and vertical and rotary in the other.

VI.

ANOMALIES OF ASSOCIATED PARALLEL MOVEMENTS.

Hypokineses. Paresis and Insufficiency of Associated Parallel Movements. *Paralysis of the lateral associated movements* of the eyes is not infrequent in destructive cerebral disease, particularly apoplexy, the site of the lesion in paralysis of dextroversion being in the left, and in par-

alysis of sinistroversion in the right half of the brain. In pure cases of this sort the power of convergence is retained, showing that the internal rectus, although incapacitated as far as consentaneous action with the opposite externus is concerned, is not disabled from performing work in conjunction with its fellow internus. This shows that in these cases we are not dealing with a conjoined paralysis of one externus and one internus, but with an inhibition of one particular *movement* of the two eyes.

Paralysis of sursumversion (without any involvement of the lateral movements) and *paralysis of sursumversion and deorsumversion together* have been observed (Gowers; Sauvineau, Trans. of VIII. Ophth. Congress, Edinburgh, 1894).

Except as diagnostic signs of the condition causing them these associated deviations are of little clinical importance. Whether there are slighter grades of these disorders, not dependent upon severe structural disease and possibly productive of more symptoms on their own account, has not, as far as I know, been positively demonstrated.

Certain cases, however, described by Savage (Oph. Record, Jan., 1896) under the name of Asthenic vertical orthophoria in which the combined up-and-down movements of both eyes (sursumversion and deorsumversion) were performed insufficiently and with difficulty would seem to belong in this category; and possibly the explanation of the conditions called by Stevens Anatropia and Catatropia may be had by assuming a weakness of deorsumversion in the former case and of sursumversion in the latter, so that in either instance both visual lines are off the proper level (see Section IX).

Hyperkinesis. Spasm of Associated Parallel Movements. Spasm of the associated lateral movements producing deviation of both eyes to the right (Spastic dextroversion) or to the left (Spastic sinistroversion) is not infrequently observed as the result of irritative lesions of the brain. Deviation to the right is produced by disease of the left side of the brain and *vice versa*.

Spasm of associated movements (particularly, combined vertical and lateral spasm, producing oblique deflections of both visual lines) also occurs as a transient and paroxysmal manifestation in epilepsy and hysteria.

The curious case described by Frost (Trans. Ophth. Soc. United Kingdom), in which there was spastic deviation of both eyes down and to the right, but in which there was no actual impairment of the movements of either eye by itself, was apparently hysterical in character.

The following peculiar case of *alternating spasm of associated parallel movements* was apparently choreiform in character and would seem to form a sort of connecting link between cases of tonic spasm, such as those just mentioned, and true nystagmus.

Choreiform Spasm of Associated Oblique Movements. Simon N., aged 13 years, came under my care Nov. 22, 1892. Chorea two years ago. Anæmic. Now shows every few minutes, especially when watched, sudden darting movements of both eyes upward and to the right and back again. The return movement takes place in two phases, the eyes shooting down and to the left somewhat beyond the primary position, and then by a sharp jerky motion coming up again to the latter. If the gaze is directed to the left, both eyes dart upward and to the *left* and then back again. In either case, whether moving up and to the right or up and to the left, the direction of the movement is at an angle of 45° with the horizontal, and the eyes are carried up until the pupils are three-quarters buried beneath the upper lids. Slight choreiform twitchings of the face accompany the movements.

Orthophoria. No diplopia. Associated movements, although sluggish, perfectly normal.

V. R. $^{15}/_{15}$ L. $^{15}/_{70}$. Under homatropine, R. + 1.00 D. $^{15}/_{15}$; L. + 1.50 D. Cyl. Ax. 85° T. $^{15}/_{30}$.

Three weeks treatment with arsenic and hydrochloric acid effected no improvement. On Jan. 15, 1893, R. + 0. 75 D. Sph., L. + 1.50 D. Cyl. Ax. 85° T. given. Patient then passed from observation and has not been seen since.

Except for the long interval between the separate spasmodic movements, the appearances presented in this case differ in no respect from those observed in nystagmus. Its development, however, in a patient with fair sight and normal muscles, and the past history and present evidences of chorea led me to regard it not as a true nystagmus. If nystagmus at all, it was a very rare form of the affection.

Parakineses. Tremor of Associated Parallel Movements (Nystagmus). Nystagmus, as ordinarily met with, consists of an equal and parallel movement of the two eyes, both executing a series of rapid oscillations in the same direction and at the same time. These oscillations, which vary in frequency from 30 to 150 a minute, occur in two

phases, a rapid darting movement first taking place in a certain direction and this being followed by a movement of return to and beyond the original position of the eyes. The latter or second phase, although slower than the first, is evidently an active movement and not a mere relaxation of the muscles that have just been spasmodically contracted. The direction of the movement is usually from side to side *(Horizontal nystagmus)*, sometimes rotary, both vertical meridians revolving in the same direction *(Rotary nystagmus)*, and very rarely vertical *(Vertical nystagmus)*. Not infrequently combinations of two, or even of all three forms are observed *(Mixed nystagmus)*.

Movements of this character point to a pathological condition of the association-centres, causing the discharge of alternate and excessive stimuli from the latter instead of the simultaneous and equal stimuli of moderate intensity, which enable the normal eye to remain steady in the position of fixation.

The eyes ordinarily, when looking at an object straight ahead of them, are kept in place by simultaneous contraction of the opposing muscles; *i. e.* they move neither to the right nor to the left, because they receive equal and simultaneous impulses from the centre for right-handed movements (dextroversion) and that for left-handed movements (sinistroversion).

If, however, these impulses, instead of being simultaneous, are alternate, so that the eyes first receive an impulse from the centre for dextroversion and then one from the centre for sinistroversion, and if these impulses alternate rapidly, we shall have the picture of *horizontal* nystagmus.

A similar want of simultaneity in the discharge of impulses from the centres for sursumversion and deorsumversion will produce a *vertical* nystagmus. And a *rotary* nystagmus will result from the discharge of alternate instead of simultaneous stimuli from the centres* which produce rotation of both vertical meridians to the right and to the left respectively. Finally, a combination of some or all of these anomalies accounts for the various forms of *mixed* nystagmus.

This conception of the *central origin* of nystagmus, and more particularly of its origin from some lesion of the different association-centres, seems forced upon us, not only

*Inferred to exist from this very symptom. The right-rotating centre, acting by itself, would produce simultaneous contraction of the right inferior oblique and inferior rectus and of the left superior oblique and superior rectus; and the left-rotating centre would similarly produce contraction of both inferior muscles of the left and both superior muscles of the right eye.

by the character of the movements executed, but also by a great variety of pathological data. For further information as to the latter, reference may be made to the exhaustive articles by Graefe (Graefe-Saemisch and Arch. für Ophth., xxiv. 3, 1895).

VII.

ANOMALIES OF CONVERGENCE.

Hypokineses. Paralysis and Insufficiency of Convergence. Absolute loss of power of convergence with retention of the power of making associated parallel movements has been occasionally observed. Since either eye can still be turned inward when acting with its fellow to move to the right or to the left, these cases cannot be referred to paralysis of the interni, but indicate some lesion of the convergence-centre, causing a **paralysis of convergence** (*Parallel squint* of Schneller).

Cases of this sort in which the paralysis was total or practically so (convergence near-point at from 2 to 6 metres), have been described as well as others in which the paralysis was marked but not complete. I myself have seen one case of the sort.

The paralysis is frequently associated with paralysis of accommodation and in some of Parinaud's and Sauvineau's cases was accompanied by paralysis of sursumversion and deorsumversion. One peculiar feature is that the prism-convergence (*i. e.* the power of overcoming prisms, base out) is retained in some of the cases (Straub).

The cases hitherto reported are:

Schweigger, Klin. Untersuch, über das Schielen 1881 (cited by Straub) 2 cases.

Parinaud, Arch. de Neurologie, 1883 (cited by Sauvineau).

Sales, Trans. Ophth. Soc. United Kingdom, iv. (1884), p. 390 (cited by Gowers).

Parinaud, Brain, ix. 330, 1887 (cited by Straub), 5 cases.

Stölting and Bruns, Arch. für Ophth. xxxiv. 3, 1888 (cited by Straub).

Benzler, Deutsch. militär-aerztl. Zeitschr., xviii. (cited by Straub).

Peters, Centralbl. f. pract. Augenheilk., xiii. (cited by Straub).

Sauvineau, Trans. VIII. Internat. Ophth. Congress, Edinburgh, 1894.

Straub, M., Arch. of Ophth., xxv. 3 (1896).

Hayne, H. W., ib.

Graefe's 10 cases (cited by Straub) come rather under the head of convergence-insufficiency than actual paralysis.

Much more frequent and important are those cases in which there is simply a greater or less weakness of convergence (**Convergence - insufficiency**). A. Graefe, who regarded these cases as quite rare, gives an excellent exposition of some ef the physical signs of the condition in a paper read before the Seventh Ophthalmological Congress (1888). In their entirety these *signs* may be stated as follows:

For distance. Lateral orthophoria or slight exophoria (1°-2°) by all tests (screen, parallax and phorometer). Associated lateral movements practically normal. Associated vertical and oblique movements often restricted (frequently in an asymmetrical manner) and field of binocular single vision often* markedly limited in some one direction above or below, indicating a weakness of an elevator or depressor (particularly the superior rectus). Diverging power (abduction) not over 9°, frequently subnormal (5°-6°). Prism-convergence (so-called adduction) often, although not always, restricted, being acquired with difficulty and performed with effort.†

For near. Marked exophoria by all tests (*i. e.* exophoria of 7° or over by both phorometer and parallax, and noticeable deviation out behind the screen). Associated lateral movements normal. Pc (near point of convergence) 3″ or over from the root of the nose, and maintenance of eyes in position of extreme convergence for more than a moment difficult or impossible. Convergence near-point about the same whether the object is approximated from a point to the right or from a point to the left of the median line. If,

*In about 20 per cent. of the cases that I have observed. In these cases there is often hyperphoria for distance in the primary position.

†That is, the primary adduction (*i. e* the greatest amount that the patient can be got to do at the first trial) will be only 8° or 12°, and the addition of even 1° or 2° to this will produce insuperable diplopia. Moreover, the exertion of overcoming a prism of even this amount is associated with a sense of strain, and the patient, if he does succeed in overcoming the prism for a moment, cannot hold the images together. These features constitute a marked difference from those obtaining in normal eyes which, though they may have difficulty in learning to overcome prisms base out, will do so with ease and with rapidly increasing facility after a few trials.

when the convergence near-point is reached, one eye be screened and then the object of fixation is carried still closer to the nose, the uncovered eye will, although with difficulty, turn in still further, in order to follow the object, while the eye behind the screen will diverge by an equal amount (preservation of associated adduction, failure of convergence-adduction.—Graefe).

Graefe's test indicates that the convergence is relatively weak—weak, that is, as compared with the absolute power of the eye to move inward (associated adduction). But whether this indicates an *absolute* weakness of convergence or not depends upon whether we regard the associated adduction (ability of the eye to move inward while its neighbor moves outward) as always normally less than the convergence adduction (ability of the eye to move inward while its neighbor is also moving inward). Graefe proceeds upon the assumption that this is the case; but in many persons, whom I examined and who had apparently normal eyes, the associated adduction seemed the greater of the two. In such the fact that they responded to Graefe's test would not necessarily indicate the existence of anything abnormal,

As to its *nature*, convergence-insufficiency may be either accommodative or non-accommodative. **Non accommodative insufficiency** may be due (A) to *direct weakness of the interni*, *e. g.* that obtaining after a complete tenotomy of the latter, especially when the operation has been done upon faulty principles, as to relieve a divergence-insufficiency not associated with excess of convergence. This post-operative weakness, which may give rise to a very troublesome asthenopia lasting for several months, is, to be sure, not really an example of convergence-insufficiency, being really a traumatic paresis of the interni, but in its symptoms and course it so much resembles the former as to be most conveniently considered in connection with it.

Convergence-insufficiency again occurs (B) *secondarily to a divergence-excess*—this being an example of that compensatory action, already alluded to, by virtue of which a deviation, which is at first marked for one distance only, tends to become generalized so as to become apparent for all distances alike. In these cases there is at first a marked exophoria for distance accompanied by excessive diverging power (10° or over), but with only moderate exophoria for near, good converging power, and no recession of the near

point of convergence. Later, the exophoria for near increases markedly (without there being necessarily any increase in the exophoria for distance), the convergence near-point recedes, and the converging power gets to be performed with more and more difficulty. From some few observations that I have made I think it likely that this sort of extension of exophoria may occur chiefly in young persons as a result of the processes of growth, which, as is well known, favor the development of a divergence of the visual lines and which may abrogate a convergent strabismus or convert a case of parallelism of the visual axes into one of divergent squint.

If this explanation is correct these cases are instances of a gradually developing bilateral insufficiency of the interni, rather than a real insufficiency of convergence.

A similar explanation—viz., the gradual production of an anatomical divergence by developmental processes taking place during the period of growth—may perhaps account for the genesis of some of the cases of convergence-insufficiency not complicated with a divergence-excess.

Another variety (C) of non-accommodative convergence-insufficiency appears to be directly dependent upon an *insufficiency of an elevator or depreesor muscle*, and particularly of the superior rectus. This connection, in view of the assistance that these vertically acting muscles give in effecting adduction, seems not unnatural and, at all events, appears to be quite frequent. Thus in 21 successive cases occurring in my practice 6 exhibited marked insufficiency, or actual paresis of the vertical muscles, and in another series of 27 that I examined 5 at least were thus affected.

In three or four other cases there was more or less weakness of the superior and inferior recti of both eyes, causing a concentric limitation of the field of single vision. This weakness, from its varying character, could not be regarded as due to paresis, but was (D) simply one of the evidences of the *general lack of muscular power and muscular co-ordination* that these patients presented. Such cases, therefore, appear to occur especially in neurasthenia. In the latter condition, at any rate, and in allied conditions of general enfeeblement, convergence-insufficiency is of

frequent occurrence and often occasions a troublesome and intractable asthenopia which disappears only when the causal affection has been removed.

Accommodative convergence-insufficiency consists in the development of a marked divergence for near points due to non-use of the accommodation. Accommodation being usually associated with convergence, any condition which prevents the discharge of accommodative impulses will likewise tend to inhibit the convergence. Theoretically this inhibition should occur in all cases, but practically it occurs in a minority only. These cases may be classified as follows:

(a) Patients with *uncorrected myopia*, in whom there is but little necessity for using the accommodation at all.

(b) In *hypermetropes*, who having all along accustomed themselves to using their accommodation without employing a corresponding convergence acquire thereby a relative insufficiency of the latter function, which comes to light as soon as their hypermetropia is corrected.

Thus a man with a hypermetropia of 2 D. has been accustomed when looking at an object 13″ off to use an accommodation of 5 D.; but when his hypermetropia is corrected by glasses, he suddenly finds that at this distance he has to use only 3 D. of accommodation, an amount with which he has been wont to associate a much smaller degree of convergence. Not being able at first to accustom himself to these new conditions, *i. e.* not being able at once to converge to a point at 13″ without using 5 D., his visual lines diverge. This divergence often gives rise to considerable trouble (persistence of asthenopia, etc.) and constitutes one of the chief reasons why convex glasses are not tolerated by many hypermetropes.

For a similar reason convergence-insufficiency may develop (C) in *presbyopes* who first put on convex glasses for near.

The marked increase in a convergence-insufficiency generally produced by (D) the continuous use of *prisms*, base in, is also probably in part due to accommodative inaction; the use of such glasses by favoring divergence tending to relax the accommodation still more and hence to superinduce a still further failure of the convergence.

The *course* of a convergence-insufficiency varies greatly. Many cases, particularly those dependent upon general muscular and nervous weakness remain about the same

for a long time and then improve as the causal condition improves. Such cases may also show recurrences, when for any reason there is a new deterioration of the vital forces. Cases also of accommodative convergence-insufficiency due to a readjustment of the optical conditions under which the patient has been working (application of convex glasses for hypermetropia and presbyopia) usually get well, the patient accustoming himself after a while to his new refractive state. This is, however, by no means always the case, and, if a tendency to convergence-insufficiency already exists, it may become aggravated and be made permanent by the use of glasses—indeed, the development of an actual strabismus divergens may be thus superinduced, particularly when the glass chosen for a hypermetropic child has been somewhat in excess of the true hypermetropia.

Convergence-insufficiency due to tenotomy also generally tends to disappear, although this again cannot be taken as the invariable rule.

Other cases of convergence-insufficiency, and particularly those occurring in young persons and dependent upon an insufficiency of the elevators and depressors, tend to increase. This seems to take place by the development of a divergence-excess in accordance with the law of compensation already several times referred to, by which a non-comitant deviation tends to become comitant. The course of such cases, if unchecked, is first increase of the exophoria for near, second development of exophoria for distance also, with increased diverging power (Divergence excess), next the appearance of an actual divergent strabismus for near with considerable exophoria for distance (Periodic squint), and finally divergent strabismus for both distance and near (Constant squint). This tendency to a constantly increasing divergence may be favored by the injudicious use of convex glasses and particularly by the use of prisms base in, which almost always tend to make the exophoria greater and greater.*

*I observed one marked case of this sort, occurring in a girl of nine, who at first had 3° of exophoria for distance and over 6° for near, but who after using prisms for a year had for distance exophoria of 8° with abduction of 10°-12° and spontaneous crossed diplopia, and for near exophoria of 15°. Prism-convergence (adduction) 0°.

As has been seen, convergence-insufficiency may be *complicated* with an insufficiency of one of the vertical muscles, the latter affection, indeed, in these cases probably being the cause of the former. In other cases, a divergence-excess is present which is sometimes the cause, but more often the effect, of the convergence-insufficiency. The latter may also, particularly in those whose muscular system generally is weak, be complicated with a divergence-insufficiency.* Slight cases of this sort, in which the insufficiency of divergence is not great enough to produce esophoria for distance constitute the asthenic exophoria of Savage.

The *symptoms* of convergence-insufficiency are asthenopia, either simple, or associated with headache and pain in the eyes, conjunctival irritation, and spontaneous diplopia, producing blurring of vision for near work. Asthenopia is a pretty constant symptom, being met with in all the varieties, although in my experience more apt to occur in the non-accommodative than in the accommodative form. It is often very marked and may incapacitate the patient from doing near work.

The symptoms are by no means necessarily in direct relation with the amount of the deviation, being, in fact, more pronounced in deviation of medium degree than in those which have assumed the proportions of a regular squint.

It is for this reason, probably, that the use of concave glasses in myopes affected with convergence-insufficiency sometimes causes distress; since these glasses tend to increase the impulse to convergence and thus reduce a large deviation, which is insuperable and gives no trouble, to a smaller one which can be overcome by effort and hence gives more annoyance. On the other hand, when the deviation is very small to begin with, the symptoms may be aggravated by the use of convex glasses and of prisms base in, which tend to make it larger and therefore more troublesome.

The *treatment* of convergence-insufficiency must aim first at *removing the cause* of the trouble. Hence in neu-

By exercise of the convergence with prisms the exophoria for distance was reduced to 0°-3°, that for near to 5°-6°, the prism-convergence was raised to 40°-50°, and there was no more spontaneous diplopia. This improvement was maintained up to the time that the patient was last seen (7 months after the treatment had been discontinued).

*I have notes of a case of this sort in a myope of 8-10 D., in whom there was an esophoria of 14-20° (i. e. a real convergent squint) for distance, with diverging power of 2-3°, and exophoria of 8° for near.

rasthenia general strengthening measures (out-of-door exercise, tonics) are indicated and are for the most part successful. Accommodative convergence-insufficiency requires the careful correction of the refraction in myopia, while in hypermetropia frequently an under-correction will be indicated. Indeed, it is a safe general rule to fully correct myopia and to under-correct hypermetropia whenever much exophoria exists; and in presbyopia under the same conditions to give a weaker convex glass than would otherwise seem indicated.

The immediate causal indication requires *training of the convergence*, which can, in general, be effected by systematic exercise with prisms base out. This often gives strikingly good results, but in some cases fails altogether.

The wearing of *prisms* base in should be resorted to only as a temporary expedient, on account of their tendency to produce increase of the trouble; and these prisms should be discontinued at once as soon as signs of such an increase begin to manifest themselves.

Finally, if an *operation* is decided upon, advancement of the interni, coupled, in case a divergence-excess is present, with a tenotomy of the externi, should be made. Tenotomy of the externi alone seldom gives any lasting result.

Hyperkineses. Spasm and Excess of Convergence. Marked tonic **spasm of convergence**, so that both eyes are turned strongly inward, has been observed in hysteria.

Minor degrees of over-action of convergence (**Convergence-excess**) are of frequent occurrence. They may be either accommodative or non-accommodative in character.

The *physical signs* of a convergence-excess not complicated with a divergence-insufficiency are as follows:

For distance. Orthophoria or moderate esophoria (1°-3°) by all tests (phorometer, screen and parallax). Associated lateral movements normal. Diverging power normal or but slightly subnormal (5°-8°). Prism-convergence (adduction) normal in amount, rapidly acquired, and easy to produce and maintain. Exercise of convergence not infrequently causing the development of temporary homo-

nymous diplopia. Homonymous diplopia sometimes also producible by will and apparently without effort.

For near. Esophoria by all tests, often exceeding that for distance.* Convergence near-point $1\frac{1}{4}''$-$1''$ or less.

Non-accommodative convergence-excess may be either (a) idiopathic or may (b) be secondary to a condition of divergence-insufficiency. The former appears to be rare, although I have met with two or three cases. The secondary variety, on the other hand, appears to be quite common, and develops according to the compensatory law by which deviations in general, tend to become equalized for distance and near. In this case, of course, there will be marked esophoria for distance and the diverging power (abduction) will be much reduced.

Non-accommodative convergence-excess probably comprises most of the cases described by Savage under the name of sthenic esophoria.

Accommodative convergence-excess (Accommodative esophoria, Accommodative convergent squint, Pseudo-esophoria) is very frequent. It occurs under the following conditions:

(a) *Uncorrected hypermetropia.* The effect of this in producing inward deviation of all degrees from a slight esophoria to a marked strabismus is too well known to require further comment here.

(b) In *myopes* who for the first time use a concave glass for near points. Such patients will add to the natural amount of convergence for the point they are looking at (which convergence they have hitherto been accustomed to make without using any accommodation) the extra convergence imposed in sympathy with the accommodative effort that they now make for the first time.

(c) In *presbyopes* (particularly, hyperopic presbyopes) at the beginning of the presbyopic period. These patients have to exert a very strong effort in order to stimulate their flagging accommodation to the point necessary for distinct

*Unless, however, the patient really fixes upon the test object, the esophoria for near may vary greatly and even be replaced temporarily by exophoria. This is particularly apt to be the case in accommodative convergence-excess, where the amount of hypermetropia may be such as to prevent the patient from seeing the object distinctly.

vision, and in sympathy with this excessive call made upon the accommodation an excessive effort of convergence is simultaneously made.

(d) *As the result of the instillation of a mydriatic.* The increase of a convergent squint by the instillation of atropine was observed by Long and Barrett (Ophth. Hosp. Reports, xii. 1888–1889), who found that it occurred in 11 cases out of 38 in whom this mydriatic was employed. These cases, however, had been under the influence of the atropine for several days. In cases which are but recently under the influence of the mydriatic an increased tendency to convergence due to the latter appears to be even more frequent. Under these circumstances esophoria may develop where orthophoria existed before, and a pre-existing esophoria of moderate amount may develop into a well-marked convergent squint.

The correct explanation of this phenomenon was first given by Savage, although I myself came independently to the same conclusion. The convergence here is evidently due to the excessive effort which the patient makes to see distinctly under the unusual conditions presented by mydriasis. Not being aware that he cannot accommodate, he makes a violent effort to do so, and in making this effort sends out a correspondingly strong impulse for convergence. The ciliary muscle does not respond, but the interni do; and, as the impulse was excessive, they respond by producing an excessive convergence. The condition, in fact, is quite analogous to the excessive secondary deviation of the sound eye when its fellow attempts to perform fixation by means of a paralyzed muscle.

As under the mydriatic the sight is more blurred for near than for distance, this fruitless effort to see distinctly by attempting to put into motion a paralyzed accommodation will be more excessive, and hence too the esophoria will be more pronounced, for near points. For distance, the esophoria will generally be greater in proportion to the degree of hypermetropia and the consequent blurring of sight, and will often disappear altogether as soon as the refraction is corrected. These facts are shown in the following cases:

Esophoria. Large increase under mydriatic. Miss K., aged 20. Refraction (under homatropine) + 0.25 sph. ⊃ + 0.50 cyl. ax. V. each. When not under mydriatic shows esophoria $^3/_4°$ for both distance and near (with and without correction of refraction); diverging power, 6° Under homatropine, without correction of refraction, esophoria for distance over 8°, for near over 15°, diverging

power 8°. Under homatropine, with correction of refraction, esophoria for distance 5°, for near over 15°.

2. J. B., male, aged 15. Hypermetropia (homatropine) 0.50 D. Before homatropine, esophoria 3° for distance, 0° for near. Under homatropine, (with and without correction of refraction), $2\frac{1}{2}$° for distance, 7° for near. Diverging power 4°.

3. Kath. S., aged 32. Hypermetropia 2.25 D (under homatropine). Before homatropine, esophoria 2°-3° for distance, 1° for near; diverging power 5°. Under homatropine, without correction of refraction, 12°-20° for distance, 12° for near (*i. e.* has an actual convergent squint). Under homatropine and with correction of refraction, esophoria $\frac{1}{2}$° for distance; little, if any for near.

The *course* in case of convergence-excess is very variable. The accommodative variety, in particular, often decreases or disappears spontaneously either as a result of developmental changes, favoring the genesis of a divergence, or in consequence of the decrease of the hypermetropia, or because the patient gives up the accommodative effort and with it the effort to converge. Its usually speedy and total disappearance in cases coming under categories B, C, and D above given, is to be ascribed to the last mentioned cause.

In other cases, particularly in the very young who are beginning to tax their accommodation more and more with school work, the deviation increases; the regular course being, first, increase of the esophoria for near, then increase of the esophoria for distance with reduction of the diverging power (Development of divergence-insufficiency), next the development of an absolute squint for near points where accommodative effort is most required (Periodic squint), and lastly strabismus convergens marked for both distant and near (Constant squint). A squint when thus fully developed usually remains permanently, but may disappear in latter life through the agencies mentioned in the preceding paragraph.

As already noted, convergence-excess may be *complicated* with divergence-insufficiency, the latter condition being either the cause or the effect of the former. It is also frequently complicated with some form of vertical deviation, producing hyperphoria in addition to the esophoria. It seems, in fact, not unlikely that these vertical de-

viations play an important part in the genesis of the excessive tendency to convergence.

The *symptoms* of convergence-excess are asthenopia, headache, and spontaneous homonymous diplopia, with sometimes more marked reflex disturbances. These symptoms, however, are much less pronounced and constant than in cases either of convergence-insufficiency or divergence-insufficiency, and when present are usually due to the attendant hypermetropia or, at least, disappear when the latter is corrected.

The *treatment* of convergence-excess is largely causal, consisting particularly in the correction of the refraction. In doing this we shall do well to follow the rule that when there is marked esophoria and, particularly, when there is more esophoria for near than for distance, we must fully correct any hypermetropia and, on the other hand, undercorrect any myopia, that may be present.

The result of treatment, both as regards the removal of the deviation and the relief of the symptoms are very good. Even in well-marked convergent squint a cure is effected much oftener than is generally supposed, the only requisites being that the refractive treatment should be kept up long enough (a year at least).* Long and Barrett (l. c.) analyzing the results in 102 cases, found that a complete cure was effected in 37, while out of 61 cases under 10 years of age 27 (or 44 per cent.) were cured.

In addition to correcting the refraction we may try to break up the excessive tendency to convergence by abolishing the accommodation altogether for a time. This we effect by keeping the eyes under *atropine* for a number of days or even several weeks. This may also, if Long and Barrett's figures hold good generally, be used as a means of prognosis, for these authors found that of 5 cases which were improved by atropine all were subsequently improved by glasses, while of 6, in whom the deviation was not affected by the atropine, only 3, and of 4, in whom the atropine made the convergence worse, only 1 received any relief from the correction of their refraction.

*This, becomes the continuous relaxation of the interni thus produced allows the weakened externi to act to greater advantage and finally to regain their tone (Long and Barrett).

Exercise of the divergence with *prisms* is of no service in this condition, and the wearing of prisms base out is to be deprecated as tending to perpetuate and increase the deviation instead of curing it.

If these means fail and an *operation* is thought advisable on account of the deformity or the symptoms, tenotomy of the interni may be done, combined, in case a divergence-insufficiency is present, with an advancement of the externi.

VIII.

ANOMALIES OF DIVERGENCE.

Hypokinesis. Divergence-Insufficiency. Weakness of the diverging power (Divergence-Insufficiency) is characterized by the following signs:

For Distance.—Esophoria of varying amount (usually 2°–8°), by all tests (phorometer, screen, and parallax). Associated lateral movements normal. Diverging power very much reduced, the reduction being often proportionate to the degree of the esophoria (*e. g.*, with an esophoria of 3° or 4°, the diverging power is about 4°, and with an esophoria of 5° or 6°, the diverging power is only 2°). In the typical cases of this anomaly, however, the diverging power is disproportionately low, being, for example, only 2° or 3°, when the esophoria is 1° or 2°, and being, perhaps, only 3° or 4° when there is orthophoria or actual exophoria for distance. Exercise of divergence with prisms, base in, is sometimes associated with a sense of decided muscular strain.

Prism-convergence (adduction) normal, or often somewhat deficient. Exercise of the convergence often gives rise to a temporary homonymous diplopia, and the latter may also in some cases (particularly when the diverging power is much reduced) occur spontaneously, or be evoked by simply placing a red glass before one eye.

For Near.—Signs, unless the condition is associated with a convergence-insufficiency, or with a convergence-excess, fairly normal (*i. e.*, slight esophoria or exophoria,

and convergence near-point at about the proper distance).

In its *origin* divergence-insufficiency is either idiopathic or secondary to a convergence-excess.

Uncomplicated *idiopathic* divergence-insufficiency is comparatively rare, and, moreover, some of the cases that would seem to fall in this category are probably either really secondary to some disorder of the vertical muscles (elevators or depressors), or are examples of a spurious divergence-insufficiency, *i. e.*, of a condition in which there is actual weakness of the externi themselves, either natural or produced by operation. In cases of the latter kind the divergence is indeed weak, but the power of making lateral movements is also lessened, and hence these cases do not fairly come under the head of a simple impairment of the diverging function.

Complicated idiopathic divergence-insufficiency is more common. In these cases there is either a convergence-insufficiency, which has developed simultaneously with the disorder of divergence, or there is a convergence-excess, which is the result of the latter.

Secondary divergence-insufficiency develops in the manner already described (see Section VII.) from a progressively increasing convergence-excess. Cases of this sort might be confounded with those of the foregoing category, *i. e.*, those in which a primary divergence-insufficiency is followed by excessive convergence action. The distinctive points between the two may be stated as follows:

PRIMARY DIVERGENCE-INSUFFICIENCY, WITH SECONDARY CONVERGENCE-EXCESS.	PRIMARY CONVERGENCE-EXCESS, WITH SECONDARY DIVERGENCE-INSUFFICIENCY.
Esophoria for distance moderate (not over 8°), and may be less than the deficiency in the diverging power (abduction).	Esophoria for distance often great, and when small usually disproportionately large in comparison with the deficiency of diverging power.
Esophoria for near small and usually less than that for distance.	Esophoria for near usually greater than for distance.
Condition stationary.	Condition often progressive.

The *course* pursued by a divergence-insufficiency is indicated in what has already been said. The idiopathic variety shows but little tendency to change, the amount of

esophoria and of divergence-weakness often remaining the same for years. This variety frequently becomes complicated with a condition of convergence-excess, which, however, also remains of moderate degree. On the other hand, a divergence-insufficiency, which is secondary to a convergence-excess, is often progressive, the advance continuing in many cases until a moderate deviation is converted into a marked and constant strabismus convergens.

The *symptoms* of a divergence-insufficiency are often very troublesome. Asthenopia as a result of near work is not so much complained of, unless there is a simultaneous convergence-insufficiency, but headache and other more remote reflex pains, a sense of constriction in the head, stomach disturbance, general inertness and lassitude, and even interference with the general nutrition are often met with. Spontaneous diplopia may also occur, being, naturally, more marked for distance than for near. One peculiarity in the symptoms is that headache and pronounced asthenopic sensations (feeling of strain and tiring in the eyes), together with a sense of confusion and dullness in the head, are especially apt to be produced by looking long and intently at distant objects, particularly when moving or when brightly illuminated. Such symptoms are hence often occasioned by watching a theatrical performance, a ball-match, a procession, or a moving throng of people. (Panorama-asthenopia, or panorama-headache of Bennett.)*

The *treatment* of divergence-insufficiency presents many difficulties, the condition being intractable and the result uncertain.

Direct exercise of the divergence with prisms, base in, has not, in my experience, been of the least avail. Exercise by the performance of systematic lateral movements of the eyes seems unphysiological, inasmuch as it is the externi that are thus practiced, and not the function of divergence *per se*, and, moreover, in these movements the interni are practiced along with the externi. Some, however, seem to have obtained good results by this method.

The constant wearing of prisms, base out, is in most

*Annals of Ophthalmology, January, 1897.

cases a dangerous expedient, as tending to cause disuse and a consequent further enfeeblement of the abducting power.

If an operation is resorted to, it should, in the idiopathic cases at all events, be an advancement of the externi rather than a tenotomy of the interni. The latter operation by itself is almost always nugatory, the condition tending, after a temporary period of improvement, to return to its original state. Moreover, the tenotomy, if thorough enough to be efficacious, is liable to substitute for the unpleasant asthenopia for distance an almost equally annoying asthenopia for near. In the secondary cases, however, or wherever a marked convergence-excess is present, tenotomy of the interni may be performed; but even then it is preferably combined with advancement of the externi.

Hyperkinesis. Divergence-Excess. Over-action of divergence *(Divergence-Excess)* is marked by the following signs:

For distance. Exophoria, usually marked (from 4° upward) with noticeable deviation out behind the screen. Associated lateral movements normal. Diverging power excessive (over 9°), the excess over the normal amount of 6° or 8° being often roughly proportioned to the degree of the exophoria, but sometimes being disproportionately larger. Prism-convergence (adduction) in uncomplicated cases normal, although possibly performed with difficulty at first. Crossed diplopia for distance often occurring spontaneously, or producible at will.

For near. Conditions, unless a convergence-insufficiency also exists, fairly normal (*i. e.*, exophoria less, or, at all events, not much greater, than for distance, and near point of convergence about in its normal situation).

A divergence-excess may either be primary in *origin* or be secondary to a convergence-insufficiency.

Primary divergence-excess occurs not infrequently as an uncomplicated affection, but still more often is associated with a vertical deviation (which may really stand in genetic relation with it), or with a consecutive converg-

ence-insufficiency, It may also be associated with a convergence-excess. I have observed several instances of this latter combination, in which, with exophoria for distance and a high diverging power, there was also excessive power of convergence, and either actual esophoria for near, or at least an exophoria slight in amount, and much less than that for distance.

The development of a *secondary* divergence-excess from a convergence-insufficiency has already been traced (see Section VIII). These cases, like the simple ones of convergence-insufficiency which represent only a less advanced stage of the same process, often show complicating insufficiency of the vertical muscles. Indeed, as before remarked, this last-named condition would seem to constitute the real cause of the divergence which takes place, first for near and then for distance.

The differential points between a primary divergence-excess with a secondary convergence-insufficiency and a primary convergence-insufficiency with consecutive divergence-excess are as follows:

PRIMARY DIVERGENCE-EXCESS WITH SECONDARY CONVERGENCE-INSUFFICIENCY.	PRIMARY CONVERGENCE-INSUFFICIENCY WITH SECONDARY DIVERGENCE-EXCESS.
Exophoria about equal for distance and near.	Exophoria for near much greater than for distance.
Converging power and prism-convergence not excessively affected.	Converging power greatly affected.
Recession of convergence near-point moderate.	Recession of convergence near-point marked.
Condition shows little tendency to progress.	Often markedly progressive.

As above stated, cases of primary divergence-excess show but little tendency to progress. I have had one such case under observation for over nine years, in which the divergence, although large, has shown but moderate fluctuations—indeed, may be said to have remained practically unchanged—during the whole time. Cases, on the other hand, of secondary divergence-excess are often progressive, a moderate degree of exophoria developing gradually into a marked divergent squint.

The *symptoms* presented by a case of divergence-excess are frequently slight. The most troublesome are those due

to an associated convergence-insufficiency, and, if this is absent, the patient may complain of nothing except possibly of a spontaneously occurring crossed diplopia for distance, or of the deformity occasioned by the noticeable deviation of the eyes. Headache and asthenopia may, however, also occur.

The *treatment* of a divergence-excess will be addressed mainly to the correction of the refraction and to the relief of an associated convergence-insufficiency. If an operation is performed, tenotomy of the externi may be done; but it will often have to be supplemented by systematic exercise of the convergence and even by advancement of the interni.

IX.

ANOMALIES OF SURSUMVERGENCE.

Hypokinesis. Sursumvergence-Insufficiency. It is not certain whether sursumvergence, *i. e.*, the separation of the visual lines in a vertical plane, is ever performed by the eyes in the execution of normal movements. It seems, however, likely that some such action may be serviceable incidentally in the act of elevating or depressing the eyes. If so, great impairment of the sursumvergence would constitute a serious deficiency. As far as I have seen, however, limitation of sursumvergence appears to have no special injurious effect.

Those who regard the amount of sursumvergence as a measure of the strength of the elevators and depressors (which it surely is not) lay more stress than I have done on the limitation of this function, and recommend systematic exercise of the latter by means of prisms, base up or down, in cases where it is subnormal. I have had some experience with these methods, but not enough to enable me to speak with assurance as to their value. My results, such as they were, have not led me to expect much from training of this sort.

Hyperkinesis. Sursumvergence-Excess. Excessive power of sursumvergence is observed in many patients affected

with hyperphoria and particularly in myopes who have an artificial hyperphoria from wearing ill-fitting concave glasses. The latter, as they shift in various positions, produce prismatic effects changing in degree and in direction, and hence requiring a variable effort to overcome the diplopia to which they would naturally give rise. This constant exercise gives rise often to a considerable increase in the power of sursumvergence, the latter sometimes attaining 10° or more.

A true *sursumvergence-excess*, *i. e.*, a state of habitual divergence of the visual lines in a vertical plane, due to excess of sursumvergence action, constitutes one of the varieties of hyperphoria, and probably accounts for some of the cases of strabismus sursumvergens and strabismus deorsumvergens. These cases must not be confounded with a similar vertical deviation due to paresis or insufficiency of some of the individual muscles. The differential diagnosis between the two will be established by making the screen and diplopia tests in different portions of the field of fixation, when, if there is an insufficiency of any individual muscle, a deviation or a diplopia, showing a characteristic increase in some one direction of the gaze, will be found to exist (see Section VI).

In sursumvergence-excess either the right or the left visual line may be habitually higher than the other (*right* and *left hyperphoria*), or each alternately may be higher (*alternating hyperphoria*). If the deviation is considerable and not habitually overcome, the condition is known as a vertical squint (*hypertropia*), which again is called right or left hypertropia, according as the right or the left visual line is above. Vertical squint is also classified into *strabismus sursumvergens* when the lower eye is the one that habitually fixes, and *strabismus deorsumvergens* when the upper is the fixing and the lower the non-fixing eye.

Regularly, in such cases, the deflection follows the law of associated movements. That is, if the right eye deviates up behind the screen when the left eye is fixing, the latter will, when the screen is shifted so as to cover it, move down in company with the right, which now, being

uncovered, is descending in order to get into the position of fixation. In certain remarkable cases, however, the *deviation of both eyes* is up *(anatropia)*, or of both eyes is down *(catatropia)* behind the screen. That is, if the right eye deviates up behind the screen when the left is fixing, the latter, as soon as the screen is shifted and the right eye moves down to get into the fixing position, will move up. In this case the visual lines, instead of remaining at a constant angle, as in the case before cited, will approximate until they become parallel, and will then diverge in the opposite direction.

Stevens *(Annales d'oculistique*, CXIII., 3, April, 1895,) was the first to carefully study these cases and call them by the names above given.[1] His view of them is that they are due to the fact that initial elevation or initial depression of both visual lines *(i. e.*, an excessive sursumversion or deorsumversion) is, in these anomalous cases, the natural state, to which each eye tends to return when not used for vision. But the condition may also, and perhaps more plausibly, be ascribed to a spasm of sursumvergence, or rather of the action opposed to sursumvergence (that, namely, by which the visual lines, when vertically diverged, are brought together again).

X.

ANOMALIES OF ROTATION-MOVEMENTS.

Anomalous conditions, in which divergence or convergence of the vertical meridians of the two corneæ (other than the physiological divergence which occurs when the visual lines are converged) have been described by Savage. His views, however, although urged with much plausibility, are still far from being demonstrated. It seems likely that these conditions, if they exist at all, are rare and of comparatively little significance.

[1]Schweigger, to be sure *(Arch. f. Augenheilk.*, XXIV., 3-4, 1894), mentions such a case, but without attempting any explanation of the phenomenon.

XI.

RECAPITULATION.

The conditions that I have sketched embrace all the different deviations, both manifest and latent, that are usually described. As the system of classification here proposed differs radically from that generally employed, it may be well, in a brief recapitulation, to show the points of contact between the two.

The ordinary classification divides deviations into Outward (or divergent), Inward (or convergent), and Vertical (upward or downward).

These deviations may be due to the following causes:

SUMMARY OF DEVIATIONS.

I. Inward Deviations (Esophoria, Convergent Strabismus) may be due to:

a. Over-action of one or both internal recti or of the other adductors of the eye (superior and inferior recti).

b. Under-action of the external rectus or of the other abductors (the obliques).

c. Under-action of the center for producing divergence movements (divergence-insufficiency).

d. Over-action of the center for producing convergence movements (convergence-excess, which, in turn, may or may not be due to excessive accommodative action).

e. Two or more of the above causes combined.

II. Outward Deviations (Exophoria, Divergent Strabismus) may be due to:

a. Under-action of the internal rectus or of the other adductors (superior and inferior recti).

b. Over-action of the external rectus or of the obliques.

c. Under-action of the center for producing convergence movements (convergence-insufficiency, which, in turn, may or may not be due to insufficiency of accommodative action.)

d. Over-action of the center for producing divergence movements (divergence-excess).

e. Two or more of the above causes combined.

III. Upward and Downward Deviations (Hyperphoria, Strabismus Sursumvergens and Deorsumvergens) may be due to:

a. Over-action of an elevator or depressor muscle.

b. Under-action of an elevator or depressor muscle.

c. Both of the above causes combined.

d. Sursumvergence-spasm, either uncomplicated or combined with under-action or over-action of an elevator or depressor.

IV. Mixed Forms (Hyperphoria combined with Exophoria, Hyperphoria combined with Esophoria, and Esophoria in one part of the field of view combined with Exophoria in another) are frequent.

The *differentiation* of these separate groups included under the above general heads is readily made, for all have well-marked signs which are detailed in the foregoing sections (V.–VIII.). The diagnosis, indeed, can in almost every case be made at once by applying the three broad principles laid down in Section IV. (at the end of the remarks upon comitant and non-comitant deviations).

The diagnostic points indicative of the chief conditions met with are recapitulated in the following table.

DIAGNOSTIC TABLE—TERMS EMPLOYED.

Pc (convergence near-point), the distance of the near-point of convergence from the bridge of the nose, when the object of fixation is carried toward the eyes in the median line.

Pc (R), the distance of the near-point of convergence from the bridge of the nose when the object of fixation is carried directly toward the patient's left eye from some point situated to the front and right of him (see Fig. 3 and accompanying explanation). Pc (R) will, in this case, denote the point at which the right eye sags off in con-

vergence. Similarly, *Pc(L)* denotes the point at which the left eye ceases to converge when the object is carried from the left side directly toward the right eye.

Convergence-adduction, the power possessed by either eye of moving inward in response to a convergence-stimulus. Its measure is the Pc.

Associated adduction, the power possessed by either eye of moving inward in performing associated parallel movements, *i. e.*, when the other eye is moving outward. It is regularly greater than the convergence-adduction.

Prism-convergence (the adduction of most) is the ability to converge the eyes, when overcoming prisms, base out.

Prism-divergence (the abduction of most authors) is the ability to diverge the eyes as measured by the degree of prism, base in, which the eyes can overcome. This is said to be *proportionate* to the amount of an exophoria or esophoria when it equals 7 + the exophoria or 7 — the esophoria (for distance).

A. OUTWARD OR DIVERGENT DEVIATIONS. — (EXOPHORIA, DIVERGENT SQUINT.)

I. Deviation and its evidences (exophoria, crossed diplopia) noticeably greater in some directions of the gaze than in others. Pc abnormally remote. Pc (R) greater or less than Pc and still more so than Pc (L). Excursion of one eye and its field of fixation abnormally increased or reduced in some one direction, both for distance and near. **Under-Action of an Adductor or Over-Action of an Abductor Muscle.**

NOTE—The diagnosis as to whether it is under-action or over-action that is present may be made from the points detailed under the head of diagnosis (4) in Section V.; and the specific diagnosis of the muscle affected may be made from the tables of diplopia in same section (6). As there stated, a crossed diplopia (or an exophoria) which increases as the eyes are carried to the right indicates weakness of *some* muscle of the left eye or over-action of *some* muscle of the right eye; and any exophoria which changes markedly in degree as the eyes are shifted can be due only to under-action of an internus or over-action of an externus.

II. Deviation and its evidences (exophoria, crossed di-

plopia) sensibly the same in all directions of the gaze. Pc (R) equals Pc (L). Excursion of both eyes and their fields of fixation normal (at least for distance).

(a.) Deviation or exophoria slight for distance. Marked for near by all tests. Pc abnormally remote. Convergence-adduction less than associated adduction (Graefe's test—see Section VI.). Prism-divergence not specially great (usually 8-10°, or may be subnormal). Prism-convergence subnormal and effected with difficulty. **Convergence-Insufficency.**

NOTE.—The diagnosis between an *accommodative* and a *non-accommodative* convergence-insufficiency will be made by reference to the etiology (see Section VI.) and by observing the effect of glasses. The latter, if suitably adjusted, usually relieve a trouble of purely accommodative origin.

(b.) Exophoria marked for distance: less for near (relations for near may be nearly normal). Pc normal, or nearly so. Prism-divergence large (in typical cases disproportionately so; *i. e.*, it is greater than 7° + the exophoria for distance). Prism-convergence often normal and effected with facility. **Divergence-Excess.**

(c.) Exophoria marked for both distance and near. Pc abnormally remote. Convergence-adduction less than associated adduction (Graefe's test—see Section VI.). Prism-divergence large, but not disproportionately so, compared with the amount of exophoria. Prism-convergence usually reduced—often greatly. **Divergence-Excess with Convergence-Insufficiency.**

NOTE.—The determination as to whether the divergence-excess is secondary to the convergence-insufficiency, or is primary, may be made by the differential diagnosis given in Section VII. This condition is not always easy to diagnosticate from a comitant deviation produced by under-action of one or both interni, combined with over-action (due to compensatory contraction) of the externi. Moreover, it is not improbable that a long-continued divergence-excess may give rise not only to an insufficiency of convergence, but to a weakness of the interni *per se*, due to their protracted inaction and to the unfavorable conditions under which they work.

(d.) Exophoria marked for distance. For near, slight exophoria, orthophoria, or esophoria. Pc normal or excessively near. Convergence-adduction greater than associated adduction. Prism-divergence large, sometimes disproportionately so. Prism-convergence normal and readily effected. **Divergence-Excess with Convergence-Excess.**

NOTE.—These cases are sometimes difficult to diagnosticate from

those of anatomical preponderance of both externi (structural deviation), combined with a convergence-excess.

The convergence-excess in these cases, being often accommodative, will frequently disappear upon the use of the proper glasses.

(c.) **Convergence-Insufficency with Divergence-Insufficiency.** (See Inward Deviations, B. II., d.)

III. **Mixed conditions,** in which the deviation increases more or less in certain directions of the gaze, but in which the phenomena noted under II. are likewise present (combination of over-action or under-action of muscles with a divergence-excess or a convergence-insufficiency); and conditions in which, by the weakness of some muscles and the over-action of others, a comitant exophoria or divergent squint has been established, are not readily analyzed into their constituent factors.

B. INWARD OR CONVERGENT DEVIATIONS—(ESOPHORIA, CONVERGENT SQUINT).

I. Deviation and its evidences (esophoria, homonymous diplopia), noticeably greater in some directions of the gaze than in others. Pc abnormally near. Pc(R) greater or less than Pc and still more so than Pc(L). Excursion of one eye and its field of fixation abnormally increased or reduced in some one direction, both for distance and near. **Under-Action of an Abductor or Over-Action of an Adductor Muscle.**

NOTE.—For the specific differentiation see Section V., remarks on diagnosis, (4) and (6). As there stated, a homonymous diplopia (or an esophoria) which increases as the eyes are carried to the right, indicates weakness of *some* muscle of the right eye or over-action of *some* muscle of the left eye; and an esophoria which changes markedly in degree as the eyes are shifted can be due only to under-action of an externus or over-action of an internus.

II. Deviation and its evidences (esophoria, homonymous diplopia) sensibly the same in all directions of the gaze. Pc(R) equals Pc(L). Excursions of both eyes and their fields of fixation normal (at least for distance).

(a.) Esophoria marked for distance; slight or replaced by exophoria for near. Pc normal. Convergence-adduction equal

to or more or less than the associated adduction. Prism-divergence low (may be disproportionately so, *i. e.*, is less than 7°—the esophoria for distance). Prism-convergence normal, or often subnormal. **Divergence-Insufficiency.**

(b.) Esophoria slight for distance; more for near. Pc excessively near (less than 1 inch). Convergence-adduction may be greater than associated-adduction. Prism-divergence normal, or but slightly subnormal (never disproportionately low). Prism-convergence normal. **Convergence-Excess.**

NOTE.—The diagnosis between an *accommodative* and a *non-accommodative* convergence-excess will be made by reference to the etiology (see Section VI.) and by observing the effect of the long-continued use of glasses (or of atropine), which in most cases will relieve a difficulty of purely accommodative origin.

(c.) Esophoria marked for distance and near. Pc excessively near. Convergence-adduction equals or is greater than associated adduction. Prism-convergence low in proportion to degree of esophoria for distance, or disproportionately low. Prism-convergence normal. **Divergence-Insufficiency and Convergence-Excess.**

NOTE.—The determination as to whether the divergence-insufficiency is secondary to the convergence-excess, or is primary, may be made from the differential diagnosis in Section VII.

(d.) Esophoria marked for distance; exophoria (of more than 4° or 5°) for near, noticeable by all tests. Pc abnormally remote. Convergence-adduction less than associated adduction. Prism-convergence low. Prism-convergence subnormal and hard to train. **Divergence-Insufficiency with Convergence-Insufficiency.**

(e.) Convergence-Excess with Divergence-Excess.
(See Outward Deviation, A. II., d).

III. **Mixed conditions,** analogous to those given under Outward Deviations III. exist, but are hard to analyze.

C. VERTICAL DEVIATIONS—(HYPERPHORIA, STRABISMUS SURSUMVERGENS AND DEORSUMVERGENS.)

I. Hyperphoria or its evidences (vertical diplopia) increasing in some one direction of the gaze.

(a.) Hyperphoria increasing as the eyes are carried upward. **Over-Action or Under-Action of an Elevator Muscle.**

(b.) Hyperphoria increasing as the eyes are carried downward. **Over-Action or Under-Action of a Depressor Muscle.**

NOTE.—For the specific diagnosis of the muscle affected see Section V., remarks on diagnosis (4 and 6).

II. Hyperphoria the same in all parts of the field (comitant hyperphoria, comitant vertical strabismus).

(a.) Due probably in most cases to under-action of an elevator or depressor, with over-action of one or more antagonistic muscle. **Mixed Cases.**

(b.) Sometimes to equal vertical divergence of the visual lines (see Section VIII). **Sursumvergence-Excess.**

CONCLUSION.

One who proposes a new classification must be prepared to defend his position by showing that it subserves some useful purpose. That this is the case with the one propounded in the preceding pages seems to me proved by the following reasons:

1. The classification is based upon physiological facts instead of mere external appearances.

2. Its divisions correspond to natural groups, distinct in nature and symptoms, and frequently requiring widely different methods of treatment.

3. The groups so made are readily distinguishable in practice by the signs they afford.

4. We can by means of the scheme here presented analyze the frequently occurring mixed forms, and from our knowledge of the nature and tendency of the component lesions determine to which of the latter our treatment shall be addressed.

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